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ANNALS
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VOL. 57 MARCH, 1948 No. 1

I

ORBITAL COMPLICATIONS RESULTING FROM LESIONS OF THE SINUSES

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AND

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That affections of the orbit are commonly due to lesions in the paranasal sinuses is generally recognized. It is emphasized by ophthalmologists and laryngologists alike and is a common meeting ground for these specialists. It is due to the close anatomic relationship between the structures. Two thirds of the wall of the orbit consist of the thin bony plates of the sinuses. In addition, the veins of the mucosa of the sinuses anastomose with the superior and inferior ophthalmic veins in the fatty cellular tissue of the orbits. These relationships permit ready involvement from lesions of the sinuses, whether due to infection, cysts, neoplasms or trauma.

The opportunity of studying a group of cases of affections of the orbit with regard to sinus lesions as a cause revealed to me that the diagnostic problem was frequently not an easy one. In some cases unsuspected and obscure lesions of the sinuses were brought

From the Department of Laryngology, Jefferson Medical College Hospital.

Read before the Eastern Section Meeting of the American Laryngological, Rhinological and Otological Society, New York, N. Y., January 16, 1948.

to light; in others, lesions outside of the sinuses were found to be causing the orbital changes, irrespective of existing sinusitis, and in some there was an absence or minimal signs of sinus involvement in spite of marked evidence of orbital complications.

The purpose of this paper is to present some of these cases, indicate some of the diagnostic problems involved, and emphasize the close cooperation required between the ophthalmologist and the laryngologist to prevent undue delay in carrying out adequate diagnostic and therapeutic measures.

The bulk of cases of orbital complications of sinus origin are inflammatory in nature, and transmission takes place in acute infection or during an exacerbation of a chronic one. The frontal and ethmoid sinuses are the main primary sites. Less frequently the sphenoid and maxillary sinuses are responsible. However, multiple involvement of sinuses so frequently occurs that it is often difficult to fix the responsibility upon any one sinus cavity.

Nasal trauma, mucoceles, pyoceles and neoplasms are less common lesions of the sinuses which may be first manifest by orbital symptoms. In these cases, if an infection is superimposed, the inflammatory reaction in the orbit can obscure the underlying lesion.

The symptomatology of orbital complications as it concerns the cases of this discussion may be grouped as follows:

1. The symptoms of inflammatory changes in the orbit.

2. The symptoms of disturbances from mechanical causes due to encroachment upon the capacity of the orbit.

3. The orbital apex or sphenoid fissure syndrome.

The symptoms of inflammatory changes due to sinus infection will vary to some extent, depending upon which sinus is involved. However, the variation really depends more upon the extent or degree of involvement of the orbital contents by the infectious process. No positive information as to the sinus responsible can be ascertained from the symptoms present. Following Hubert's[1] classification of orbital infections due to sinus infection, Imperatori and Burman[2] give an excellent summary of the symptomatology. The classification is an arbitrary one depending upon severity or extent of orbital involvement. The infections are divided into five groups:

Group 1.—Inflammatory edema of the eyelids, with or without edema of the orbit, most marked near the sinus involved. The in-

fection is limited to the sinuses and there is only inflammatory edema of the lids. The eyeball is movable in all directions and the vision is usually not affected. If the edema extends into the orbital tissue, then there appears a slight exophthalmos and some limitation of the movement of the eyeball.

Group 2.—Subperiosteal Abscess. There is infection of the bony wall and the periosteum, with a collection of pus between them. There is a circumscribed swelling, which is painful to the touch. The eyeball is displaced and there is interference with its mobility, depending upon the location of the subperiosteal abscess. The pus may extend into the lids and break through forming a fistula.

Group 3.—Orbital Abscess. The orbital tissue proper is infected by a direct extension through the orbital wall and fascia, or through the venous circulation. Exophthalmos and chemosis are marked, the eyeball is immobile and there is interference with vision. The pus may spread to the lid or the conjunctiva and break through.

Group 4.—Orbital Cellulitis. Phlebitis of the ophthalmic veins occurs when the infection extends into the orbital tissue through the venous circulation. Prostration and high temperature are prominent features. Extreme exophthalmos, chemosis, fixation of the eyeball, and disturbance of vision are noted. These cases may not show any pus macroscopically, but on microscopic examination there is evidence of phlebitis, periphlebitis and scattered areas of necrosis.

Group 5.—Cavernous Sinus Thrombosis. This complication gives essentially the picture of a severe orbital cellulitis as seen in Group 4. It has been called by Mosher[3] the anterior or orbital type of cavernous sinus thrombosis. The infection focuses in the ophthalmic veins and the orbit, in contrast to the posterior type where the major infection is in the cavernous sinus itself. Two additional signs may be present: involvement of the orbit on the side of the noninfected sinuses; edema over the mastoid emissary vein. Macewen[4] states that in more than half the cases of cavernous sinus thrombosis the process extends to the sinus of the opposite side, since the sinuses are connected with each other by the circular sinus. Positive blood culture is usually present, but this may also be the case in orbital cellulitis.

The symptoms of disturbances from mechanical causes occur with mucoceles, pyoceles, chronic hyperplastic sinusitis, cholesteatoma, cysts, and neoplasms, and are manifested by displacement and disturbance in the mobility of the globe, injury to the optic nerve, changes in refraction and disturbance in the drainage of tears.

Orbital apex or sphenoid fissure syndrome is a rare symptom complex caused by affection of the vessels and nerves which pass through the superior orbital fissure and the optic foramen. The characteristic symptoms are ptosis, complete ophthalmoplegia, impaired vision and pain corresponding to the ophthalmic division of the fifth nerve. There is little said of this syndrome[5] in American literature, but it is mentioned not uncommonly by the French. The etiology is usually considered to be trauma, syphilis, tuberculosis or a neoplasm. It can also be caused by infection in a large sphenoid sinus which extends into the greater wing sufficiently to approximate the nerves in the superior orbital fissure. Such a case is reported by Ib Kyaer[6] and another is discussed (Case 7) in this report.

Case reports, statistical studies, and reviews of the literature show that as high as 72 per cent of cases of orbital infection arise from nasal sinus suppuration.[7] An extensive literature on the subject has become available in the past decade, and when evidence of involvement of the orbit occurs during the course of acute infection of the sinuses or an exacerbation of a known chronic infection, the connection is promptly recognized. The case immediately becomes the joint responsibility of the laryngologist and the ophthalmologist, and effective treatment is instituted. However, we found in our study that difficulty and delay in diagnosis arose when:

1. The sinus infection was not apparent from symptoms or rhinologic examination. The symptoms of the original nasal infection may have cleared before the orbital signs became manifest.

2. A noninflammatory lesion of the sinuses was present but was obscured by inflammatory reaction.

3. There was a history of sinus infection and acute inflammation of the upper respiratory tract was present, but the focus was elsewhere.

4. Lesions of the sinuses, such as a mucocele, cyst or osteoma, gave no nasal symptoms and encroachment on the orbit was so gradual that eye symptoms were not manifest until involvement became marked.

Roentgen examination is the greatest single aid in diagnosis. However, the findings must be correlated with the clinical evidence. This is the province of the laryngologist or the ophthalmologist and depends upon his ability to understand the meaning of the roentgenologist's report and his understanding of the value of the films submitted.

The advent of penicillin has largely solved the problem of treatment of orbital infection due to sinusitis. Prior to penicillin the literature is largely taken up with pro-and-con discussion as to which was the best procedure: conservative, local, intranasal measures, directed to promote ventilation and drainage of the sinuses; or immediate external opening. Today penicillin in adequate dosage parenterally is accepted as the most efficacious therapeutic measure. When the infection is due to susceptible organisms, it will prevent grave complications of osteomyelitis, cavernous sinus thrombosis, meningitis and brain abscess. If these serious conditions are present, penicillin offers a good chance of cure. Formerly, they were often considered hopeless regardless of what heroic surgical measures were tried and too often drastic futile procedures were carried out on the questionable grounds that the patient was going to die anyhow. In comparing results of treatment with penicillin with those of other forms of therapy, one is amazed at the effectiveness of this agent. The customary procedures have been largely obviated, and if required, they are much more limited or conservative than was formerly considered necessary.

Penicillin is effective in infections due to gram-positive bacteria. Fortunately these organisms cause the vast majority of sinus infections, so that if penicillin is given adequately at the onset of acute infection, it is rare for complications to occur. When they do occur, we are apt to attribute the failure to improper or inadequate application rather than to the agent itself.

Treatment with penicillin has become so simple and commonplace that it has all but eliminated the laryngologist in handling sinus infections. The physician who first sees the patient may be lulled into indifference or carelessness while waiting for the magic drug to carry out its brilliant work. However, it will not affect a mucocele, pyocele, chronic hyperplastic sinusitis with inadequate nasal drainage, or none, or a new growth, and if these lesions are present, there will be an unwarranted delay in arriving at an accurate diagnosis and in instituting proper care of the sinus lesion and the orbital complication. These lesions require mechanical measures. Mucoceles, pyoceles and hyperplastic lesions must be incised and drained through the sinuses into the nose. The neoplasm must be removed completely and adequate nasal drainage established.

REPORT OF CASES

I—The Inflammatory Group—Acute and Chronic Infection in the Sinuses with Involvement of the Orbit.

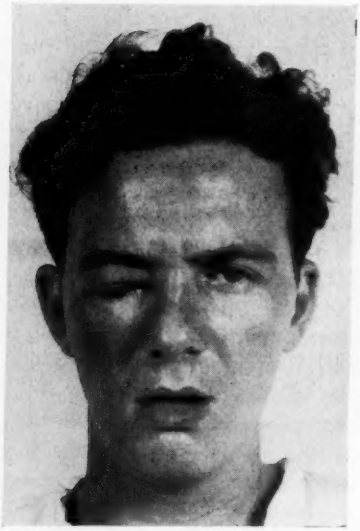

Fig. 1, Case 1.—This case represents the simplest type of orbital complication from acute sinusitis; inflammatory edema of the lid. The eyeball was movable in all directions and vision was not affected. The infectious process remained limited to the sinuses.

CASE 1.—Male, aged 20, was seen July 15, 1947, with marked swelling of right eye and pain in the right side of the face and forehead. The pain had started with dramatic suddenness 48 hours before, awakening him during the night. Twenty-four hours later the right eye became swollen. There was marked edema of the right upper lid; the eye could not be opened (Fig. 1). There was marked tenderness over the right frontal sinus and the nasofrontal area. There was purulent drainage from the right choana and the anterior part of the middle meatus. Transillumination showed cloudiness of the right frontal and maxillary sinuses. Temperature was 100.2° F.; the white blood count, 12,000; x-ray films showed cloudiness of the right frontal, ethmoid and maxillary sinuses. Treatment consisted of 100,000 units of penicillin every 12 hours given intramuscularly. One per cent ephedrine in physiologic sodium chloride solution was prescribed as nose drops every second hour. Recovery was prompt. In seven days the eye had returned to normal and there was no evidence of any residual infection in the sinuses.

Fig. 2, Case 2.—Severe orbital cellulitis and abscess. The origin of the infection could not be determined.

This case represents the simplest type of orbital complication from acute sinusitis: inflammatory edema of the lid. The eyeball was movable in all directions and vision was not affected. The infectious process remained limited to the sinuses.

CASE 2.—Male, aged 21, was seen January 7, 1941, with orbital cellulitis. There was marked exophthalmos of the right eye, redness and edema of the lids and chemosis of the conjunctiva, fixation of the globe, dilatation of the pupil and loss of vision. The infection started six days prior to admission with swelling and redness on the right side of the nose, which rapidly spread to the upper and lower lids closing the eye (Fig. 2). A small incision had been made over the region of the lacrimal sac, but no pus was found and the swelling and redness increased. Nasal examination showed no evidence of sinus infection. X-ray study of the sinuses, however, revealed clouding throughout and the absence of the right frontal sinus. The interpretation given was pansinusitis with marked changes in both maxillary sinuses. Because of the x-ray report and inability to explain the orbital cellulitis on other grounds, an exploratory operation on the right ethmoid area was done through an external incision. No evidence of any infection was found. The orbital cellulitis progressed in spite of incision and drainage of multiple abscesses, and enucleation had to be done. Following this, the infection subsided. The cause of the orbital infection was never definitely settled. Penicillin was not available in 1941, but sulfanilamide and sulfathiazole were

Fig. 3, Case 3.—Cavernous sinus thrombosis. Acute sinusitis was present, but an unsuspected furuncle in the left nasal vestibule was the cause of the orbital complication. Recovery occurred following the use of penicillin in combination with heparin.

both given and may have helped prevent spread of the infection to the cavernous sinus and the intracranial structures.

This case of severe orbital cellulitis and abscess illustrates how difficult it may be to determine what role the sinuses play in causing the infection. Did it come from a focus in the sinuses which was overlooked, or did it arise from a superficial skin infection which was not apparent?

CASE 3.—Male, aged 29, was seen May 13, 1947. Both lids of the left eye were markedly swollen, especially the upper, which was red and tender, with severe tenderness over the supraorbital region. The conjunctiva was chemotic, edematous, and extended between the lids (Fig. 3). The eyeball was proptosed and fixed. The pupil was dilated and fixed. The media were clear. The disc was edematous, as was the retina, which had a milk-white color. The eye was blind. Temperature was 105° F. On May 8, 1947, five days prior to admission, he developed an acute cold. Two days later he had severe chills and fever which he thought might be due to malaria from which he suffered while in the army. The next day the left upper lid was swollen and the eye painful. Twenty-four hours later pain was very severe and vision was lost.

There was a history of repeated attacks of left-sided sinusitis. In view of this, along with onset during an acute cold, the x-ray report indicating cloudiness of the left antrum and ethmoid sinuses, our first impression was that we were dealing with severe orbital cellulitis and cavernous sinus thrombosis secondary to sinus infection. However, subsequent examination revealed a furuncle with a surrounding indurated area on the outer wall of the left vestibule, of which the patient made no complaint. The intranasal indications of acute sinus infection were not marked, so it is believed that the furuncle rather than the sinus infection was the cause of the orbital involvement.

The day after admission there was swelling of the lids of the right eye.

The culture from the furuncle showed staphylococcus aureus. The blood culture was negative.

Treatment consisted of 100,000 units of penicillin every third hour for the first 24 hours. This was then changed to heparin, 200 mg. in 100 cc. P.S.S., administered over 24 hours at the rate of 12 drops per minute, and penicillin, 200,000 units every sixth hour in .5 cc. physiologic sodium chloride solution given subcutaneously. The penicillin was increased five days later to 200,000 units every fourth hour, at which time clinical and x-ray signs of bronchopneumonia developed.

Heparin was continued for ten days. The prothrombin levels varied from 71 to 86 per cent (80-100 per cent normal). There was no bleeding. The patient recovered but did not regain vision in the eye. He left the hospital on the twenty-sixth day. Marked edema of the conjunctiva of the lower lid persisted for some weeks.

This case is of interest because it illustrates that even in the presence of an acute sinusitis the sinus infection may not be the focus of the orbital complication. In this instance an unsuspected furuncle in the left nasal vestibule was the cause. It also illustrates the importance of penicillin in combination with heparin as a therapeutic agent in this grave disease.

CASE 4.—Female, aged 65, was examined November 12, 1947, because of purulent discharge from a fistula at the outer third of the left supraorbital rim. A probe could be passed through the fistula into the frontal sinus. The upper lid was pulled up at this point by scar tissue, and the eye could not be closed to cover the cornea properly. There was also present a conjunctivitis, opacity of the

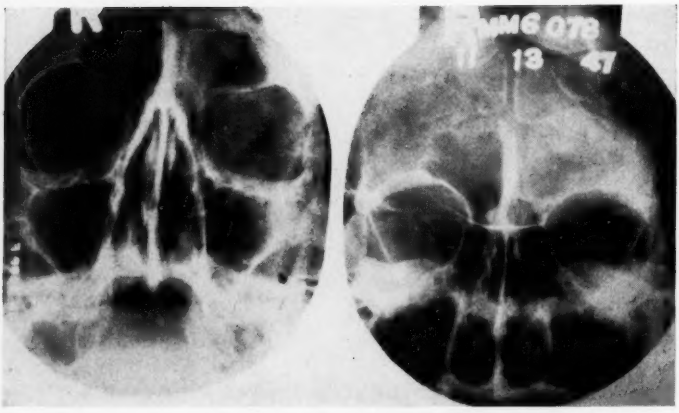

Fig. 4, Case 4.—Roentgenograms showing cloudiness of the left frontal sinus with thickening of the walls and a cancellous structure adjacent to the cavity, indicating condensing osteitis about the sinus.

inner half of the cornea and marked impairment of vision of the affected eye. Culture of the pus showed staphylococcus aureus. She had no pain, no headache, and no nasal symptoms. Nasal examination revealed a moderately large polyp attached to the anterior tip of the middle turbinate, but there was no evidence of inflammation or discharge.

The fistula had been present about 18 months and followed incision of a large painful swelling which had closed the eye. She had been given penicillin and x-ray therapy to bring about healing. At the time of the incision a polyp had been removed from her left nasal chamber, but nothing else had been done to her nose. The swelling above the eye came on very gradually over a period of years and was painless at first. However, a short time before it was incised it became very painful, red and tender. Incision of the mass was followed by discharge of pus and relief from pain.

X-ray films showed cloudiness of the left frontal sinus and thickening of the walls and a cancellous structure adjacent to the cavity (Fig. 4). These findings indicated condensing osteitis about the sinus cavity and chronic inflammation within. A site of dehiscence could be determined by x-ray examination.

Under intratracheal anesthesia the frontal sinus was approached externally by a curvilinear incision just below the supraorbital rim which was carried down the side of the nose to expose the ethmoid area. A defect was found in the bony floor of the frontal sinus, measuring 1.5 x .5 cm., oval in shape, the margins of which were atrophied to knife-like thinness. The lining membrane of the sinus was markedly thickened and intact except for the fistula at the outer angle. The sinus was a moderately large, single cavity. The osseous walls were thickened and had a "pebbled" appearance. There was no nasofrontal duct. A probe could be passed from the nasal chamber through a large anterior ethmoid cell up to the frontal sinus, but this cavity was sealed off. The lining membrane of the ethmoid cell appeared normal. There was no indication of ethmoid disease. The thickened membrane of the frontal sinus was removed in one piece. The histologist reported pseudostratified, ciliated, columnar and transitional epithelium, covering edematous, vascular tissue containing plasma cells and lymphocytes.

A large opening was made from the frontal sinus into the nose by way of the large ethmoid cell without removing the membrane lining of the wall. It will serve as a nasofrontal duct. The external wound and the fistula were closed and they healed by primary union.

In this case the invasion of the orbit began with the appearance of a swelling above the eye and was due to a herniation of hyperplastic mucosa of the frontal sinus through an erosion in its floor. It was primarily an inflammatory orbital tumor of sinus origin without intranasal signs. No pain was present until the development of osteitis and a subperiosteal abscess. The true clinical picture was probably not recognized earlier because of the absence of intranasal signs of infection. The establishment of adequate nasal drainage at the time of the original incision would have prevented the eye complications.

CASE 5.—Male, aged 36, was admitted October 9, 1946, complaining of progressive swelling of the right eye of six weeks' duration, associated with right supraorbital and nasofrontal pain. For two years he had been subject to episodes of swelling and lacrimation of the right eye with pain lasting several days. No disturbance of vision was complained of. No previous history of sinus disease was obtainable. For three months he had had a foul, brownish postnasal discharge.

Examination revealed a firm bulging mass beneath the right supraorbital ridge, filling the right nasofrontal angle. A bony de-

hiscence was palpable in the supraorbital ridge. Proptosis of the right eye, ptosis of the upper lid, and slight limitation of outward rotation with diplopia were noted. Intranasal examination showed polypoid degeneration of the right ethmoid cells and mucopurulent discharge from this area.

Roentgen examination showed erosion and depression of the right supraorbital wall with slight bone sclerosis. The supraorbital ridge was eroded at its medial half.

Through an external curvilinear incision the right frontal and ethmoid sinuses were opened. A cyst containing heavy mucopurulent material was found arising from a frontally placed ethmoid cell, extending laterally to the supraorbital notch and posteriorly the full length of the ethmoid labyrinth. No bony structure of the ethmoid labyrinth remained; it had been replaced by cystic necrotic polypoid tissue. The frontal sinus itself was not greatly encroached upon, but its membrane was thickened. Two areas in the superior orbital plate were eroded with dural exposure.

This case also comes under the clinical classification of inflammatory or pseudo-tumors of the orbit which result from chronic hyperplastic inflammatory changes in the ethmoid or frontal sinuses. The invasion of the orbit was caused by erosion of the orbital plate by a pyocele associated with polypoid hyperplastic chronic ethmoid disease. In this instance the relationship between the sinus infection and the orbital symptoms was indicated by the intranasal evidence of ethmoid disease. Removal of the necrotic polypoid tissue was necessary to cure the condition.

CASE 6.—Male, aged 72, was seen August 4, 1946, when admitted to the hospital because of severe pain in the right eye and the right frontal area, double vision and impaired vision in the right eye. The pain began one week prior to admission; the eye symptoms, three days. An acute cold and bronchial cough had been present for two weeks. There was ptosis and weakness of the right upper lid. The eyeball was fixed in the straight ahead position with questionable proptosis. The media were clear, the discs normal; the pupils were round, regular, and reacted normally to light and accommodation. Tension was normal and the eyeball not tender. Diplopia was present in all fields, and vision reduced to 5/200 in the right eye. He had complete ophthalmoplegia on the right side due to involvement of the second, third, fourth, and sixth nerves. The intranasal examination showed nothing remarkable. The right frontal and maxillary sinuses were dark upon transillumination; the left, clear.

Fig. 5, Case 6.—Roentgenogram showing cloudiness of the very large sphenoid sinus.

This clinical picture has been referred to as the orbital apex or sphenoid fissure syndrome, and is the result of impairment of the nerves passing through the superior orbital fissure and the optic foramen.

X-ray films revealed marked clouding of the right maxillary, ethmoid, and sphenoid sinuses; less marked clouding of the frontal. The bony structure of the right posterior orbital wall appeared indistinct. The optic foramen showed no abnormalities, but the superior orbital fissure could not be clearly outlined. The left sinuses were normal. The sphenoid sinus was very large (Fig. 5).

The diagnostic problem was whether the eye symptoms were due to a retrobulbar lesion such as aneurysm or neoplasm, or a sinusitis with a nonsuppurative inflammatory swelling within a very large sphenoid, causing pressure upon the nerves passing through the superior orbital fissure.

Response to penicillin, 25,000 units every three hours intramuscularly, indicated that acute infection within the sinuses was the cause of the eye symptoms. The pain disappeared in 48 hours, the ptosis of the upper lid was less, and there was some up-and-down movement of the eyeball. He continued to improve and the eye returned to normal. The x-ray film showed clearing of the sinuses coincident with the clinical improvement, and the superior orbital

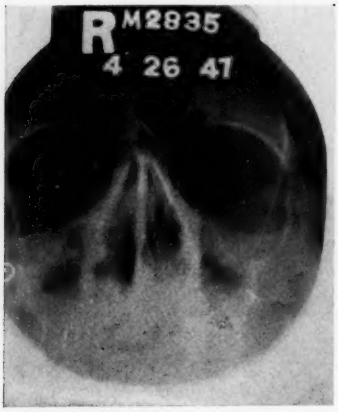

Fig. 6, Case 7.—Roentgenogram of sinuses which indicated destruction of the orbital plate of the right ethmoid labyrinth, with loss of cellular detail of the right ethmoid cells.

fissure was clearly outlined. Penicillin was continued for three weeks. Eighteen months later there was no trouble with the eye or the sinuses.

This case illustrates the orbital apex or sphenoid fissure syndrome resulting from inflammation in a very large sphenoid sinus. An interesting feature was the absence of exudate. The nerve involvement must have been due to pressure from the edematous lining membrane of the sinus. If a dehiscence existed in the osseous wall, it would make this more probable.

II—Mucoceles of the Frontal and Ethmoid Sinuses.

CASE 7.—Male, aged 32 years, was seen May 5, 1947. He was referred for examination of the sinuses because of an x-ray report of ethmoiditis. He had already recovered from an acute orbital infection under penicillin therapy. X-ray studies of the sinuses indicated destruction of the orbital plate of the right ethmoid labyrinth, with loss of cellular detail of the right ethmoid cells (Fig. 6). There was diplopia on right lateral gaze, and limited movement of the globe to the right. The nasal chambers, nasopharynx and pharynx appeared normal. The right antrum was dull upon transillumination and when irrigated by puncture through the inferior meatus,

about a fluid dram of pus was obtained. The infection in the antrum cleared following the placing of a window under the inferior turbinate. He left the hospital following this operation June 3, apparently well. He reported to the eye department a month later because of increased diplopia, failing vision, and proptosis. Vision was 20/20 in the left eye and 20/90 in the right. He was readmitted with a diagnosis of mucocele of the ethmoid sinuses. It was learned at this time that following a truck accident in May 1946, in which he suffered an injury to his head and loss of consciousness, his right eye appeared to be more prominent than his left.

An external ethmoidectomy was done under local anesthesia, which revealed the presence of a large cystic formation filling the entire ethmoid labyrinth, none of the cellular compartments remained. The orbital surface of the cystic formation was adherent to the orbital fascia. Medially it reached the middle turbinate and posteriorly the sphenoid. It was filled with heavy, yellow, mucoid material. It was diagnosed as an infected mucocele. It was removed, the middle turbinate being left in place. Healing occurred without complication and the eye returned to normal.

This was at first thought to be an ordinary case of orbital infection similar to Case 1, except that it was more advanced. In addition to the edema of the lids, the eyeball was displaced and its mobility impaired. The symptoms came on suddenly during an acute upper respiratory infection, and there was pus in the right antrum. However, the primary cause of the orbital symptoms was the mucocele of the right ethmoid cells, which probably dated back to his injury. The sudden development of orbital signs was due to inflammatory reaction in the mucocele resulting from the acute sinusitis.

CASE 8.—Male, aged 48, was first seen January 9, 1947, complaining of right frontal headache radiating upward and over the right hemicranium, which occurred for the first time in November 1946. It was described as a dull, constant, throbbing pain, relieved by lying down. Two months after the onset, ptosis of the right eyelid became evident but there was no diplopia.

Examination of the patient revealed fullness of the right upper lid and a palpable rounded prominence in the right nasofrontal angle corresponding to the floor of the right frontal sinus. This yielded on pressure and was slightly tender. All other examinations of the eye and nose and the neurological examination were negative. Roentgen examination showed an old linear fracture through the right

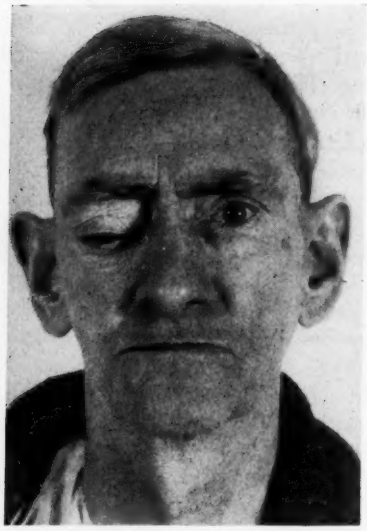

Fig. 7, Case 9.—Mucocele of a right supraorbital ethmoid cell causing proptosis of the right eye with downward and lateral displacement.

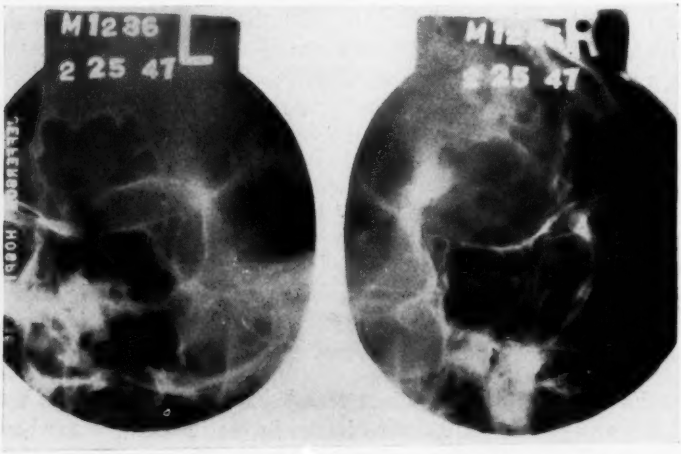

Fig. 8, Case 9.—Roentgenograms of the sinuses showing increased density of the wall of the right frontal sinus, with erosion of the greater part of the roof of the orbit.

parietal and temporal bones. This was caused by a fall at the age of 12 years, resulting in marked deafness. The frontal sinuses were small; the contour of the right frontal was smooth and enlarged by erosion. This was believed to be a mucocele.

Two operative procedures were carried out. The first consisted of opening the anterior frontal plate, removing very heavy, grayish, mucoid material, and instilling iodized oil into the enlarged frontal sinus which encroached upon the orbit. The iodized oil demonstrated a blocked nasofrontal duct. The second procedure consisted of a right external ethmoidectomy to relieve the nasofrontal obstruction. It was not necessary to remove the posterior ethmoid cells nor the middle turbinate.

This case illustrates a typical mucocele of the frontal sinus, the cause of which may have been trauma. A large part of the frontal sinus wall had become atrophic and bulged into the orbit because of pressure, ultimately becoming absorbed. The lining mucous membrane was atrophic and had largely disappeared. There was an absence of inflammatory reaction. He had been studied by the ophthalmologist, fitted with glasses, and subsequently referred to a neurologist before a nose and sinus investigation was sought. Cure was obtained by establishing adequate nasal drainage.

CASE 9.—Male, aged 64, was first seen on February 24, 1947. He complained of loss of vision and prominence of the right eye. Drooping of the right upper lid developed first in the spring of 1943, then double vision, and finally complete loss of vision. The eye was operated upon two years prior to admission, without improvement. There had been no headache, no pain, and only slight discomfort over the right eye. There was no history of any nasal symptoms at any time. Examination showed the right eye proptosed with downward and lateral displacement; the upper lid could not be elevated (Fig. 7); and all extra-ocular movement was limited, particularly on upward gaze. Palpation of the medial and superior angles of the right orbit revealed the presence of a slightly tender, soft, cystic swelling. No erosion of the supraorbital ridge was palpable. The right optic disc showed flaring of its margins with one or two diopters' elevation. Roentgen studies showed increased density of the wall of the right frontal sinus with fuzziness of its outer borders (Fig. 8). There was erosion of the greater part of the roof of the orbit. An external approach by means of a curvilinear incision over the ethmoids corroborated the diagnosis of mucocele. A cyst-like formation taking its origin from a supraorbital

ethmoid cell was found. It had expanded laterally between the orbital plate and the capsule to erode all of the floor of the right frontal sinus and much of the superior orbital plate to the extent that the dura was exposed at the lateral and the posterior aspect of this plate. The frontal sinus cavity was not entered appreciably by the cyst and its membrane appeared normal. The anterior ethmoid cells were encroached upon. Postoperatively the patient's course was satisfactory with the return of vision to normal.

This is a case of a mucocele involving the right ethmoid capsule which developed gradually over a period of four years.

III.—Epidermoid Cysts of the Frontal Bone. This interesting condition is not primarily a lesion of the sinuses, but it is presented here because in our case it offered a diagnostic problem in that clinically and roentgenologically it had many of the features of a mucocele of the frontal sinus.

CASE 10.—Female, aged 21, had symptoms beginning in September 1944 of drooping of the eyelid and diplopia. When first examined April 30, 1946, she complained of pain and numbness over the right eye of one week's duration. Examination of the right eye revealed ptosis of the upper lid, choked disc, proptosis, exophthalmos, and hypalgesia corresponding to the first division of the trigeminal nerve. She was subject to episodes of profuse nasal discharge of chocolate-colored material. Sneezing caused momentary proptosis of the right eye. Examination of the nose and throat was negative. Palpation in the region of the upper right palpebral-orbital fold showed some fullness, tenderness and roughening of the supraorbital ridge. Upward rotation of the right eye was limited, and its vision poor. Roentgen studies showed an intracranial soft tissue mass just lateral to the right frontal sinus and extending posteriorly in the anterior fossa. The entire roof of the orbit and the lateral wall of the right frontal sinus were eroded. The mass was surrounded by a large multilocular air cavity (Fig. 9).

The neurosurgeon explored this cavity by reflecting a skin and bone flap. A large amount of dark, inspissated material which looked like wet cornflakes was present, lying free in the cavity and the frontal sinus. This material, grossly and histologically, had the appearance of cholesteatoma. Most of the cavity was air containing. There was erosion of the posterior plate of the frontal bone. The dura and the brain were pushed posteriorly. The frontal sinus was affected only to the extent that its cavity had been communi-

Fig. 9, Case 10.—Roentgenograms showing an intracranial soft tissue mass, just lateral to the right frontal sinus and extending posteriorly into the anterior fossa. The mass is surrounded by a large multilocular air cavity.

cated with by this large cyst-like cavity. The nasofrontal duct was patent and the lining membrane normal in appearance. The final diagnosis in this case was epidermoid cyst of the frontal bone, with secondary erosion into the frontal sinus. The communication of the epidermoid cyst with the frontal sinus, and thence the nasal chamber, explained the collection of the air in the cavity, giving it the characteristics of a pneumocele. As the lining membrane of the frontal sinus did not appear abnormal and a normal appearing nasofrontal duct provided good drainage into the nose, it was not disturbed.

Except for the patent nasofrontal duct, discharge of brownish fluid and protrusion of the eyeball upon sneezing, this case had many of the clinical and x-ray features of a mucocele of a frontal sinus which contained air. However, a mucocele is a closed distention of a nasal sinus in which the lining membrane is atrophic or has disappeared altogether. When it discharges into the nose, it is cured. The final diagnosis depended in this case upon the microscopic examination of the material removed from the cavity.

IV.—Osteoma of the Frontal and Ethmoid Sinuses.

CASE 11.—Female, aged 51, was seen March 8, 1947, with prominence of the right eye, ptosis of the right lid, diplopia when looking to the left, and lacrimation. The symptoms had started in January 1946, with lacrimation, and progressed with proptosis, ptosis of the lid, double vision and occasional shooting pain over the right eye. Six months prior to onset of the difficulty she was accidently struck a severe blow just above the right eye with a hoe handle. It was not severe enough to cause unconsciousness or ecchymosis.

Examination revealed the right eye displaced outward, downward, and lateralward. There was a soft compressible fullness at this area above the eye and below the supraorbital ridge, with no detectable bone erosion. There was diplopia in the horizontal plane on left lateral gaze. Intranasal examination was negative. The right frontal sinus was dark upon transillumination; the left, clear.

X-ray films showed a circumscribed calcifying lesion arising from the lateral edge of the right frontal sinus and growing down upon the supraorbital region of the right eye. The mass appeared to be 1.5 to 2 cm. in diameter and to have a calcified rim. The right frontal sinus showed loss of its mucoperiosteal membrane, particularly at its lateral superior margins. An x-ray diagnosis of an unusual mucocele which had undergone calcification at the rim, with invasion of the superior portion of the orbit, was suggested.

Under local anesthesia an incision was made along the lower edge of the right eyebrow, and the mass opened. It proved to be an osteoma involving the lateral wall of the frontal sinus and invading the orbit. There was cystic dilatation of the sinus mucous membrane, with a collection of grayish mucoid material around the confines of the osteoma. The bony mass was removed and the incision closed with catgut and black silk, a small Penrose drain being left in place at the outer angle. The ethmoid and nasofrontal areas were not disturbed. She made an uneventful recovery with disappearance of the eye symptoms.

CASE 12.—Female, aged 54, was seen in the neurology clinic January 28, 1947, because of marked proptosis and almost complete blindness of the right eye. Visual acuity had been getting progressively worse for the past two years and was associated with proptosis for nine months. For 20 years she had had a bony prominence over the left frontal region which very slowly became larger.

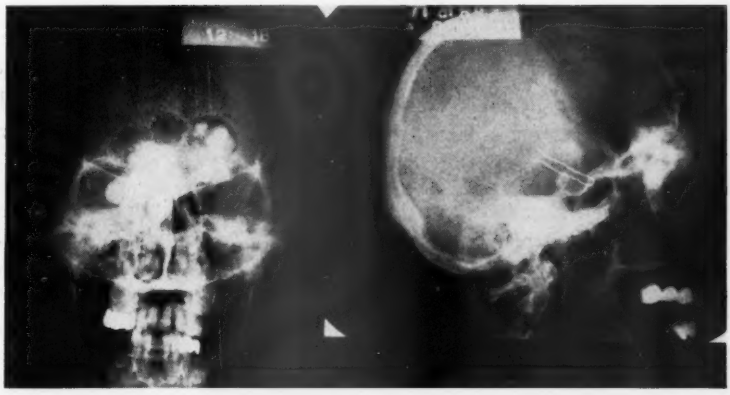

Fig. 10, Case 12.—Roentgenograms of the skull and orbit, showing multiple lobulated osteomas in both frontal and ethmoid sinuses.

Fig. 11, Case 12.—Multiple, lobulated osteoma of both frontal and ethmoid sinuses, with marked displacement of the right eyeball due to the osteoma's invading the right orbit. Prominence of the left frontal area is due to an osteoma in the left frontal sinus.

Inspection of the interior of the nose was negative. The sense of smell was lost bilaterally.

Examination of the orbits showed marked encroachment on the right orbit by a palpable bony growth, just under the supraorbital ridge. The left orbit was partially encroached upon by a similar but smaller bony growth (Fig. 11).

Roentgen examination of the skull and the orbits showed multiple lobulated osteomas in both frontal and ethmoid sinuses (Fig. 10). These osteomas were satisfactorily removed at two operations, first from the right side and nine months later from the left, through a curvilinear incision along the brow and the side of the nose. A large dural exposure was unavoidable on each side, particularly upon removing the large mass on the right side. Postoperatively in the latter instance, the patient showed some signs of meningeal irritation during the first 48 hours. Penicillin was administered before and after both operations.

Two cases of osteoma are reported not because of any diagnostic problem, as they are easily and positively identified by x-ray study. There is only one other picture that resembles it and that is of a sinus filled with lipiodol. They are reported to illustrate the unnecessary delay that frequently occurs between onset of symptoms, proper diagnosis and adequate treatment by operative removal. These tumors do not limit their growth to the path of least resistance but expand by ruthless compression in all directions, destroying everything in their path. The eye symptoms are often treated over a period of months or years as the osteoma slowly and painlessly increases in size, until it has so extensively involved the orbit and the cranial cavity that something has to be done. Removal is then difficult and hazardous. They are primarily a problem for laryngologists and should be removed as soon as they are recognized. To delay is to make a relatively simple problem difficult and dangerous.

SUMMARY

A group of cases illustrating infections, mucoceles, epidermoid cysts, and osteomas of the nasal sinuses responsible for complications of the orbit, has been reviewed. They show that the diagnostic problems involved are often complicated and that a painstaking, thorough rhinologic investigation is required to avoid unnecessary delay in making an accurate diagnosis and in carrying out adequate therapeutic measures. The laryngologist should be the first and not the

last consultant in these cases, and for him it will frequently be a problem requiring the most careful observation and study.

1830 SPRUCE STREET.

REFERENCES

1. Hubert, L.: Orbital Infection Due to Nasal Sinusitis, New York State J. Med. 37:1559-64 (Sept. 15) 1937.

2. Imperatori, C. J., and Burman, H. J.: Disease of the Nose and Throat, 2nd ed., Philadelphia, J. B. Lippincott Co., 1939, p. 242.

3. Mosher, H. P.: Discussion of Hill, F. T.: Progress in Otolaryngology Since 1937, Trans. Am. Laryng. Rhin. and Otol. Soc., 1946, p. 209.

4. Macewen: Quoted by Hajek, M.: Nasal Accessory Sinuses, 5th ed., St. Louis, The C. V. Mosby Co., 1926, vol. 2, p. 644.

5. Holt, Helen and DeRotth, A.: Orbital Apex and Sphenoid Syndrome, Arch Ophth. 24:731-741 (Oct.) 1940.

6. Ib Kyaer: Case of Orbital Apex Syndrome in Collateral Pansinusitis, Acta Ophth. 23:357-366, 1945.

7. Davis, E. D. D.: Orbital Cellulitis Due to Infection and Its Treatment, J. Laryng. & Otol. 25:834-848 (Dec.) 1937.

II

OBSERVATIONS ON BONE CONDUCTION IN FENESTRATION CASES

PHYSIOLOGICAL CONSIDERATIONS

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Very little has been published to date concerning bone conduction studies in patients with clinical otosclerosis who have undergone a fenestration operation. It is the purpose of this paper to report observations on bone conduction made during the past few years. The discussion will be divided into three parts:

1. Use of the Weber test to evaluate early postoperative hearing.

2. Audiometric bone conduction tests late in the postoperative period as compared to preoperative levels.

3. Some physiological considerations as to the possible basis of these clinical observations.

The Weber Test.—Failure to obtain a good initial improvement in hearing after fenestration is observed much less frequently now than it was a few years ago, but it still constitutes one of the major obstacles to perfection in fenestration surgery. While the so-called serous labyrinthitis is fortunately usually spontaneously reversible, we are not justified in ceasing our efforts toward its prevention or the enhancing of the reversibility of the process. Since the peak of the reaction seems to occur between the fifth and tenth postoperative days it seemed highly desirable first of all to find a means of detecting early the occasional case of severe hearing depression. When two or three weeks have elapsed after surgery, therapeutic measures are undoubtedly much less effective.

Since bone conduction tests are used to evaluate the cochlear function before surgery, it seemed that the same approach should be useful in the early postoperative period. The Weber test seemed

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From the Department of Otolaryngology, Northwestern University Medical School.

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to me to offer the simplest method of detecting an early significant drop in cochlear function. The 256, 512 and 1024 forks were used for this purpose. Of 88 consecutive cases evaluated for this study, the Weber test lateralized to the operated ear the first few days after operation in 51 instances. In the remaining cases there was no definite lateralization and in only a few was there lateralization to the unoperated ear. Before operation there was no predominance in lateralization toward either side. The tendency for more lateralization toward the operated ear after surgery must be explained on the basis of changes created by the fenestration operation.

In order to determine if the packing placed in the fenestration cavity for six days after surgery played a part in the tendency for the Weber test to lateralize more often to the operated ear, 50 unselected patients were tested in the office during their follow-up care. These patients had been operated on from six weeks to one year previously. It was found that the tendency to lateralize to the operated ear in these patients corresponded approximately to what was observed in the previous group which was tested during the hospital period. This would appear to eliminate the packing as a significant factor.

The general observation was that there was less initial gain in hearing in those patients in whom the Weber shifted to the unoperated ear four or five days following operation after having initially lateralized to the operated ear. On the assumption that hypertonic sucrose given intravenously would reduce cochlear edema and congestion, those patients who had a consistent shift of the Weber away from the operated ear were given sucrose; 200 cc. of 50% solution were given by slow intravenous drip in each of three treatments in a period of 36 hours. This tended to shift the Weber back somewhat to the operated ear in quite a few cases. Recently we have been using 50 cc. of Sorbitol instead of the 200 cc. of sucrose. Sorbitol may be somewhat more effective and has less tendency toward an occasional side reaction. Whether this reversal of the Weber would have occurred had nothing been done can, of course, not be determined with certainty at this time. However, a comparison of hearing improvement results in a group of patients so treated with a previous group of unselected consecutive cases in which no sucrose was used, would suggest that the group including the sucrose cases may have had fewer unimproved cases, particularly in the early postoperative period. It is chiefly the occasional case of early marked depression of hearing that would be expected to be benefited by such treatment.

A correlation of the shift of the Weber with early hearing improvement after surgery is shown in Table 1. Of those patients who

showed less than 15 db. gain at the six-week audiometric examination, slightly over half (50 per cent) had shown a shift of the Weber away from the operated ear. Of those who obtained a 15 db. or more gain at the six-week test, in only about four per cent had the Weber shifted away from the operated ear with no return during the hospital period. Ten patients in whom the Weber had shifted but started to return to the operated ear before their leaving the hospital all had a 15 db. or more gain. These 10 patients all had sucrose after the change was noted.

TABLE 1.—CORRELATION OF WEBER TEST DURING HOSPITALIZATION WITH AIR CONDUCTION GAIN AFTER SIX WEEKS.

	WEBER NOT SHIFTED	WEBER SHIFTED SOME RETURN	WEBER SHIFTED NO RETURN
15 db. or more gain	64	10	3
Less than 15 db. gain	5	—	6

While I am aware that the Weber test has a certain degree of fallibility, our observations to date indicate that it is a means of detecting early drop in hearing with sufficient dependability to justify its continued use for this purpose after fenestration surgery. At the present time we also take into consideration the approximate improvement in hearing as determined by air conduction fork tests at the sixth day dressing when arriving at a decision as to whether or not the ear may benefit from the intravenous administration of the solution.

In addition to the use of the Weber test, audiometric bone conduction tests were done every day during the week the patient was hospitalized after surgery. The threshold in the hospital environment was determined by testing the patient with the hospital audiometer after admission the afternoon preceding surgery. The bone conduction threshold for the ear selected for operation was established before and after operation by placing the bone conduction receiver over the zygoma and masking the other ear. The ear not to be operated on was tested in the usual manner without masking, inasmuch as the operated ear could not be satisfactorily masked after surgery. While the curve obtained in day-to-day testing gave some index as to the status of cochlear function, there were some technical sources of error which made this method not worthwhile for routine clinical testing. Perhaps the most significant finding obtained from these tests was that in quite a few instances there was a 10 to 15 db.

better threshold for bone conduction acuity immediately after operation as compared to the level existing before surgery.

Late Postoperative Bone Conduction Observations.—In view of the Weber test and early postoperative bone conduction findings, it was of interest to investigate what happened to the bone conduction threshold later in the postoperative period. Bone conduction levels for the 256, 512, 1024 and 2048 frequencies at the third postoperative month were compared with the preoperative levels of the same patients. The tests were done with masking both before and after operation. The unoperated ear would serve to a certain extent as a control for both the audiometer used and for the patient's dependability at the time of the testing. Twenty-eight cases have been tabulated for this study (Table 2). The average level for each frequency before operation is compared with the average level after operation on both the operated and the unoperated side. It will be noted that the improvement in level in the operated ear ranges from approximately 1 db. for the low frequency to 13 db. for the higher frequency. On the unoperated side the 256 and 2048 frequency levels show little change while at the 512 and 1024 frequencies the levels (thresholds) have been increased by approximately 5 db. This increase might be accounted for by the masking sound being conducted through the operated ear with sufficient intensity to interfere somewhat with the patient's hearing by bone conduction in the unoperated ear.

TABLE 2.—AVERAGE BONE CONDUCTION THRESHOLD IN DECIBELS IN 28 PATIENTS.

	OPERATED EAR			
	256 c.p.s.	512 c.p.s.	1024 c.p.s.	2048 c.p.s.
Preop.	2.2	9.0	13.8	25
3 Mo. Postop.	1.4	6.0	9.6	12
	UNOPERATED EAR			
	256 c.p.s.	512 c.p.s.	1024 c.p.s.	2048 c.p.s.
Preop.	3.5	10	11.2	25
3 Mo. Postop.	3.2	14.8	16.6	25.7

In connection with this study, it should be pointed out that, if bone conduction threshold is to be used as a basis for future evaluation of possible further cochlear nerve degeneration in fenestrated

ears, the future level should be compared with the level established after surgery and not with the one obtained before operation. Otherwise an eventual drop of 10 to 15 db. in the higher tones from the level established early after surgery might be misinterpreted as no change, if the level before operation has been used as a base for comparison.

Physiological Considerations.—It is generally believed that increased bone conduction is a frequent clinical observation in cases of conduction deafness, even though there is very little positive proof that in any given patient a conduction lesion has actually increased the hearing acuity via bone conduction. While the failure of a defective conduction mechanism to transmit extraneous masking sounds to the inner ear may account for most of the apparent increase in bone conduction acuity in middle ear deafness, this alone may not explain entirely the physiology of increased bone conduction. Individual factors such as skull resonance, bone density and the condition of the sub-aditus trabeculae all enter into determining the bone conduction threshold in each individual.

Guild[1] observed in some animal experiments that the Wever-Bray response produced by bone-conducted sound was not impaired for the 256 and 512 frequencies by removing the lenticular process of the incus. There was, however, a slight impairment on the average for the 1024 and 2048 frequencies by bone conduction. Kobrak,[2] on the other hand, obtained a slight decrease in threshold for bone-conducted sound in animals in which a conduction lesion had been produced. He used the stapedius reflex method of determining the threshold before and after the lesion was produced.

Stevens and Davis[3] in discussing the mechanism of bone conduction make the following statement:

"Experimental evidence [Bekesy[4]] appears to indicate that both air conduction and bone conduction ultimately cause similar movements of the endolymphatic fluid and of the basilar membrane in the cochlea. The air-borne vibrations arrive by way of the ossicles, whereas vibrations of the skull result in intermittent compression of the canals of the inner ear, including the labyrinth. Such compression increases the pressure in the scala vestibuli more than in the scala tympani because the round window, in communication with the scala tympani, is more elastic than the oval window, which is closed by the footplate of the stapes [Fig. 1].

"Still more important, according to Bekesy, is the fact that the semicircular canals communicate with the scala vestibuli and, when they are compressed, fluid is forced into the scala vestibuli [Fig. 1]. By a series of ingenious experiments in which bone-conducted vibrations were compensated by equal and opposite vibrations delivered to the stapes from air-borne waves and in which the freedom of movement of the stapes was altered by raising and lowering the air pressure on the tympanic membrane, Bekesy demonstrated the existence of this type of com-

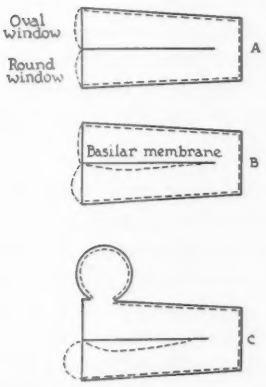

Fig. 1.—Drawing showing how compression of the inner ear by bone-conducted sound waves leads to movement of the basilar membrane. The dotted lines indicate the sizes of the various chambers and the positions of the various membranes during compression by a sound wave in the skull. (Bekesy[4])

A. Hypothetical case of symmetrical compression of the cochlea and equal yielding of the membranes of the oval and round windows. No movement of the basilar membrane would occur.

B. The round window actually yields more than the oval window to equal pressures. The basilar membrane is moved slightly toward the scala tympani.

C. The semicircular canals are compressed as well as the cochlea. Fluid forced from the semicircular canals into the scala vestibuli causes greater movement of the basilar membrane into the scala tympani.

pression. It constitutes the most important element of the mechanism of hearing by bone conduction when the external canal is open.

"When an observer listens to a tone by bone conduction and closes the external auditory canal, the tone increases in loudness. The vibration of the skull then compresses the air in the external canal and the observer virtually hears by the usual mechanism for air conduction. The loudness may then be diminished by increasing the air pressure in the external canal, thereby tensing the tympanic membrane and the ligament of the footplate of the stapes, just as in ordinary air conduction. On the other hand, when the external canal is open, tensing the tympanic membrane and ossicular chain causes an increase in loudness of the bone conducted tone, because it fixes more rigidly and prevents loss of pressure from the scala vestibuli by way of the oval window. Compression of the cavities of the inner ear and labyrinth is here the dominant factor in producing vibration of the basilar membrane."

Kobrak[5] recently reminded us that Mueller stated approximately 100 years ago that when sound approaches and passes through the

temporal bone everything moves; that is, even for ordinary air-borne conversational speech which, according to the physicists, is usually around a 50 db. intensity there must be vibrations set up in the entire temporal bone as well as in the sound conducting mechanism. The hearing mechanism as a whole is constructed so as to respond best to air-borne sound.

Herzog[6] and Krainz[7] have made some detailed observations on the relationship of phase and amplitude of the conduction mechanism as compared to that of the temporal bone itself when sound is conducted directly against the bone. They observed that the movement of the ossicular chain in response to bone-conducted sound was in a phase somewhat opposed to that of the perilymph movement produced by the same sound conducted directly to the labyrinth capsule and the perilymph by the bone of the skull. Consequently, the net result was a decrease in amplitude of perilymph movement in the scala vestibuli and the scala tympani. A lesion of the conduction mechanism would either partially or completely eliminate this perilymph amplitude cancellation effect (Fig. 2).

The exact physiology of sound conduction to the organ of Corti in a fenestrated ear still remains to be accurately determined. To begin with we must again remember that when sound vibrations come in contact with the head and ear structures, everything moves. Stroboscopic examination of the fenestrated ear into which 60-cycle sound was conducted according to the method described by Perlman[8] indicates that the movements produced by this sound simulate those which can be produced by intermittent pressure with a Siegle otoscope. These observations were made on a few patients in whom there was a marked depression of hearing, presumably due to labyrinthitis, after surgery. In other words, as the pressure wave enters the external meatus and reaches the depths of the cavity, the tympanic membrane would move with the pressure wave toward the inner ear. There was not sufficient movement visible over the fenestra to determine whether the tissue over it moved in or out with the pressure phase.

In some patients who have had fenestration operations it is possible to produce a slight fistula response by applying pressure against the central part of the pars tensa. The direction of the ocular response is opposite to that elicited when the tissue in the fenestra is pushed in gently with an applicator. This would indicate that inward movement of the tympanic membrane can somehow produce outward movement in the fenestra in some instances. Whether this relationship of movement of the flap and the tympanic mem-

Fig. 2.—Drawing with tympanic cavity and ossicular chain added to Fig. 1*C*. A compression wave transmitted through the bone produces some outward movement of the drum and ossicular chain, thereby lessening basilar membrane displacement. Pressure in the tympanic cavity would exert a slight pressure against the round window membrane and also decrease basilar membrane displacement. *EAM*—external auditory meatus; *TC*—tympanic cavity; *SV*—scala vestibuli; *ST*—scala tympani.

Fig. 3.—Drawing showing fenestration cavity and tympanomeatal flap added to figure 1*C*. A compression wave passing through the skull in part traverses fenestration cavity *A* and exerts pressure against the flap over the fenestra *F*. If inward pressure at *F* exceeds outward pressure, a reinforcement of perilymph displacement toward the round window would be expected.

brane is the rule or the exception cannot be stated at this time. The possibility has been considered that in a fenestrated ear there is a reversal of perilymph phase in relation to the phase of the air-conducted sound, but this seems unlikely.

Two simple experiments have been done in an attempt to determine whether or not this reversal could exist. First, a bone conduction hearing aid applied to a fenestrated ear that has a good level of hearing produces a slightly further improvement in hearing for speech. Second, if an air-conducted and bone-conducted pure tone of identical phase are heard simultaneously by a fenestrated ear, the air-conducted sound reinforces the apparent loudness of the bone-conducted sound. In a fenestrated ear it seems most logical that the pressure phase of bone-conducted sound displaces the perilymph toward the round window. Otherwise, according to Bekesy's conception of this point as illustrated in Fig. 1, perilymph movement would proceed to both the round window and the fenestra with a resulting decrease in displacement of the basilar membrane. The postoperative bone conduction studies already discussed indicate no decrease in bone conduction hearing, but rather on the average, an increase. What, then, prevents a decrease in perilymph displacement toward the round window in the fenestrated ear and instead produces in some instances a slight increase in response to bone-conducted sound?

If a bone conduction receiver is placed against a small solid object held near a normal ear, the loudness as perceived in that ear by air conduction is comparable in intensity to that which would be produced if the receiver were placed against the temporal bone over the antrum. In an ear that has been fenestrated, the pressure wave of a bone-conducted tone would, at the same time as the labyrinth capsule is compressed, send a compression wave into the air or the packing overlying the fenestra. This inward pressure entering the fenestra should be sufficient to equalize the outward pressure created by compression of the bony capsule and in addition in some instances actually reinforce the perilymph displacement toward the round window (Fig. 3).

An intact drum is an essential requirement in obtaining maximum hearing improvement in a fenestrated ear. As has already been pointed out, inward movement of the tympanic membrane produced by an applicator moves the tissue in the fenestra out. Whether ordinary sound pressure will move the tympanic membrane sufficiently to produce movement in the fenestra is at the present time not known. However, if sound should transmit tympanomeatal flap movement from the tympanic membrane to the fenestra, it would appear to be in a phase opposite to that produced by the same pressure wave moving directly into the fenestra. The resultant movement in the fenestra would then be an algebraic summation of these two forces. On the basis of previous discussion it would seem most likely that the tissue overlying the fenestra moves in with the pressure phase of the sound. Based on this assumption, the perforation must cause a drop in hearing for some reason other than its effect of decreasing whatever movement of the flap that may exist.

The loss of hearing caused by the perforation may be related to the part played by the tympanic membrane in modifying or dampening the intensity of that portion of the sound wave which is transmitted through the tympanic cavity to the round window. The elasticity of the tympanic membrane would offer some resistance to a sound wave and consequently absorb some of its energy and decrease the pressure exerted against the round window. A perforation would decrease the pressure differential between the fenestra and the round window and consequently reduce the amplitude of perilymph movement. Sound conducted through a small tube to within a few millimeters of the fenestra appears much louder to a patient than the same sound conducted through the same tube directly to the drum. This would be additional evidence that the drum is not the principal receptor of sound in a fenestrated ear.

At Shambaugh's[9] suggestion, I placed a small piece of sponge in the perforation of a fenestrated ear to determine the effect on air conduction hearing. The sponge produced a hearing improvement equal to that obtained by placing a patch over the perforation. A piece of sponge over the round window alone produced some improvement, but not as much for the speech tones as a complete plugging of the perforation. In this same patient sound conducted directly by a small tube to within a few millimeters of the round window through the perforation appeared louder to the patient than sound directed to the fenestra. On the patient's other ear which was also fenestrated and had obtained a 15 db. level by air conduction, the sound was perceived more loudly over the fenestra than against the drum.

Bekesy[10] has recently reported actual measurements of sound pressure differences between the oval and round windows of a temporal bone and between a lateral semicircular fenestra and round window. He found a greater pressure difference between a lateral canal fenestra and the round window than between the oval and round windows in the absence of a conduction mechanism. In a given preparation the phase difference existing for a high frequency was greater than that for a low frequency. This would explain the greater increase in bone conduction hearing for the 2048 frequency as compared to the change for the 256 frequency after fenestration. Wever[11] has pointed out the importance of phase difference of a sound wave at the lateral semicircular canal fenestra and the round window in the physiology of a fenestrated ear.

For an air-borne sound wave to produce a good displacement of the basilar membrane it must arrive at the two windows in a slightly different phase. In addition, the drum membrane must be intact in order to protect the round window somewhat from sound pressure, thereby further increasing the pressure difference between the two openings when the pressure at the lateral canal fenestra is at its peak. The factors which chiefly enter into determining this pressure differential are:

1. Resistance of the tissue over and in the fenestra in the lateral canal.

2. Elasticity of the intact tympanic membrane.

3. The angle formed by a line passing through the fenestra and round window with a line which is parallel to the direction of the incoming sound wave as it reaches the inner ear. If this angle is 90 degrees there would be little or no phase difference. The further

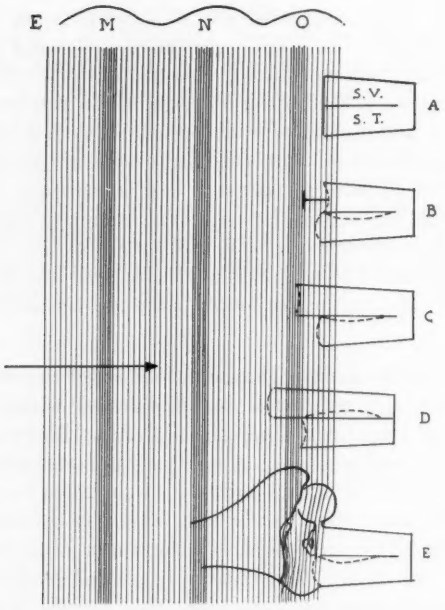

Fig. 4.—*E* represents the usual way of depicting a sound wave. The arrow indicates the direction of movement of three successive pressure phases of sound. Pressure phases of the waves are at *M*, *N* and *O*. One cycle would be from *M* to *N*, one from *N* to *O*.

SV represents the scala vestibuli and *ST* represents the scala tympani.

In the cochlea at *A*, the phase and the sound pressure are equal against the two windows and consequently there is no displacement of the basilar membrane.

At *B*, a conduction mechanism or prosthesis against the vestibular window creates a phase and pressure difference with resulting displacement of the perilymph and the basilar membrane toward the round window.

At *C*, there is a phase and pressure difference at the two windows with similar displacement.

D could represent what occurs a moment later when pressure relationship is reversed.

E is a fenestrated ear such as shown in Fig. 3, placed with the fenestra at the peak of pressure wave *O*. The tympanic membrane offers resistance to the sound pressure and increases the pressure differential between the round window and the fenestra.

Note: No particular wave length is represented in this figure. The relationship between the wave length and the cochlear length is entirely schematic.

the angle is from 90 degrees the more favorable the anatomy is to make possible a sufficient phase difference to permit good hearing by air conduction. In other words, the further the fenestra is lateral to the round window, the greater will be the phase difference. This anatomical relationship is variable and must enter into determining the possible degree of hearing improvement. There would be some tendency to symmetry in an individual's ears and the two sides would be somewhat equally favorable or unfavorable in this respect (Fig. 4).

A Pohlman[12] prosthesis should theoretically be helpful in improving the hearing in those cases in which the fenestra is not sufficiently lateral to create a satisfactory phase difference. Pohlman reports improvement in a few cases to a serviceable level by means of a properly fitted prosthesis. The relationship of the axis of the auditory meatus to the fenestra-round window line should be important. A fenestra placed in the superior or posterior vertical canal would provide less phase difference than one in the lateral canal, because the vertical canals are a few or several millimeters more medial in position. A fenestra placed on the most lateral aspect of the horizontal canal would from a physiological standpoint appear to be most favorable. In our experience the usual variations in the size of the fenestra have not appeared to be of any significance as to the degree of initial hearing improvement.

In several patients with fenestrations, I have been able to produce minor audiometric and subjective improvement in hearing by placing mercury, a tiny inflated balloon or a Way artificial drum against the tympanic membrane. The subjective improvement was particularly noticeable to those who had not quite reached the serviceable level. The improvement can be explained on the basis of dampening of sound transmission to the round window.

Since sound waves follow in general some of the principles of refraction and reflection that apply to light waves, there are undoubtedly some characteristics of size and shape of the fenestration cavity which enter into the final hearing result obtained by surgery. Would a tympanic membrane poorly visible from the meatus because of a more curved canal receive less sound pressure than one more directly in line with the outer axis of the canal? Can the size and shape of the cavity be purposely so altered that a maximum of sound pressure will reach the fenestra at the peak of the pressure phase? These are questions for which definite answers cannot be given at this time.

SUMMARY

1. The use of the Weber test is discussed as a means of helping to detect occasional cases of hearing depression in the early postoperative period after fenestration.

2. An average lowering of bone conduction threshold in fenestration cases is reported. This occurs chiefly for the 1024 and 2048 frequencies.

3. The physiology of the fenestrated ear is discussed with presentation of some evidence that air-conducted sound may enter the inner ear chiefly by passing directly through the fenestra.

4. Anatomical possibilities which may influence the maximum degree of hearing improvement obtainable by surgery are discussed.

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REFERENCES

1. Guild, Stacy: Bone Conduction. Pathways of Transmission of Sound, Annals of Otology, Rhinology and Laryngology 45:736, 1936.

2. Kobrak, H.: Tensor Tympani Muscle Reflex in Analysis of Function of Middle and Internal Ear, Annals of Otology, Rhinology and Laryngology 47:830, 1936.

3. Stevens, S. S., and Davis, H.: Hearing, Its Psychology and Physiology, New York, John Wiley and Sons, 1938, pp. 293.

4. Bekesy, G. V.: Zur Theorie des Horens bei der Schallaufnahme durch Knochenleitung, Ann. d. Physik. 14:6-10, 1932.

5. Kobrak, H. G.: Physiology of the Conduction Mechanism. Unpublished data presented before Otosclerosis Study Group, October 12, 1947.

6. Herzog, H.: The Bone Conduction Problem: Theoretical Considerations, Zeitschrift f. Hals-Nasen-u. Ohrenh. 15:300-306, 1926.

7. Krainz, W.: The Bone Conduction Problem: Experimental Results, Zeitschrift f. Hals-Nasen-u. Ohrenh. 15:306-313, 1926.

8. Perlman, H. B.: Stroboscopic Examination of the Ear, Annals of Otology, Rhinology and Laryngology 54:483 (Sept.) 1945.

9. Shambaugh, G. E., Jr.: Personal communication.

10. Bekesy, G. V.: Sound Pressure Difference Between the Round and Oval Windows and the Artificial Window of the Labyrinthine Fenestration, Acta Otolaryng. 35:301-315, 1946.

11. Wever, Glenn: Physiology of Sound Perception, Meeting of Otosclerosis Study Group, 1947.

12. Pohlman, M. E.: The Artificial Middle Ear, Annals of Otology, Rhinology and Laryngology 46:647 (Sept.) 1947.

III

THE FENESTRATION OPERATION

A SURVEY OF FIVE HUNDRED CASES

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A patient seeking advice regarding the fenestration operation usually asks three questions:

1. Am I a suitable subject for surgery?

2. What are the possible complications of such a procedure?

3. What results may I expect following the fenestration operation?

The answers to these questions will be based on experience gained from 800 operations, of which 500 were done more than six months ago.

With slight modification the original Lempert nov-ovalis technique[1] was followed in this series. The cartilage stopple,[2] the gold burr and the more recent lead burr technique advocated by Lempert were not used in this group of cases.

The operations were performed under heavy basal analgesia (six grains of barbiturate and one-fourth to one-half grain of morphine in graduated doses), supplemented by intravenous sodium pentothal administered in the ankle vein.[3] Continuous oxygen was given through an airway during surgery.

In the early cases the Lempert incision was used with the removal of a triangular piece of skin and as a result a small tympanomeatal flap was created. The mastoid cells were thoroughly removed with complete skeletization of the three bony canals of the labyrinth, making a relatively large mastoid cavity. In the later cases the single incision was used and all of the skin was incorporated in the tympanomeatal flap, thereby making it much larger so as to line the mastoid cavity

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From the Department of Otolaryngology, University of Southern California School of Medicine.

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more completely. The mastoid cells are no longer completely exenterated but are partially removed and only the horizontal bony canal is skeletinized. Very adequate exposure of the area is obtained and a small cavity is provided for epithelization by this procedure.

One per cent novocain with 1:50,000 adrenalin is injected into the tympanomeatal flap before thinning with forceps and scissors. This provides hemostasis and also balloons the tissue so perforation is less likely to occur during the thinning process. After the tympanomeatal flap is prepared, it is carefully scrutinized under magnification for any bone remnants which might stimulate osteogenesis of the fenestra after it is placed in position. A gauze pack of 1:1000 adrenalin is placed in the epitympanic area for a few moments to produce complete hemostasis before the fenestra is created. A small piece of vaseline gauze[4] is then placed over the raw surface of the flap and over the middle ear area to protect against any bone dust which might accumulate during the creation of the fenestra.

The fenestra is created in a completely dry field using neither intermittent nor constant irrigation.[5] Any accumulation of bone dust is removed by cotton applicators dipped in 1:1000 adrenalin and the polishing burr is frequently changed, thereby avoiding excessive heat over the labyrinth.

The double blue line (twin bed) technique is used to create the fenestra (Fig. 1). Using a No. 203 polishing burr, the superior blue line is made along the upper surface of the surgical dome as far anteriorly as possible. The inferior blue line is created parallel to the superior blue line and as close to the facial nerve as possible. These two blue lines with a ridge of unpolished bone between are then connected anteriorly by using a small No. 201 polishing burr. The center ridge between the two blue lines is then removed by one stroke of the larger No. 203 polishing burr. By this means the underlying endosteum is nearly always intact and the bone dust lies on the surface of the endosteum.

By using this double blue line method, trauma to the membranous labyrinth is minimized because a spinning burr is never used over the open fenestra. This is illustrated by the small incidence of postoperative labyrinthitis with secondary nerve changes noted in this series.

The edges of the fenestra are then smoothed by use of dental excavators after the method of Lempert. The vaseline gauze is removed from over the middle ear area and the tympanomeatal flap

Fig. 1.—Double blue line technique.

area and the fenestra is gently and indirectly washed by intermittent use of physiologic sodium chloride solution at body temperature.

The endosteum, which is nearly always intact after using the double blue line technique, is carefully elevated and removed, the operator using small Shambaugh hooks and working in an anterior to posterior direction. Any overlying bone dust is largely removed with the endosteum. The remainder is indirectly washed and the completed fenestra is then visualized. The tympanomeatal flap is placed in position, carefully elevated and rewashed, then replaced over the fenestra and held in place with parresined lace mesh after Lempert's original technique.

On return of the patient to bed, 1000 cc. of 5% glucose is administered and each patient receives 1,000,000 units of penicillin over a period of four days following surgery. The patient is encouraged to become ambulatory at the earliest possible date. No attempt is made to keep the head in a fixed position following surgery.[6]

Indications For Fenestration Surgery.—A patient with a progressive conduction type of hearing loss with intact ear drums and patent eustachian tubes is suitable for operation providing he does not have serviceable hearing, is in good general health and has good cochlear nerve function. Previous ear infection, or even mastoid surgery does not contraindicate fenestration, providing the ear drum is intact.

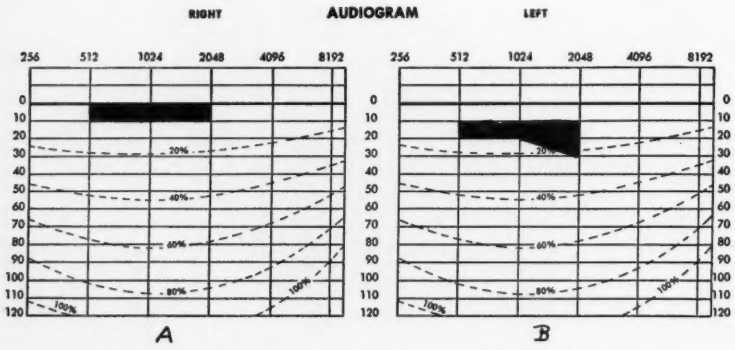

Fig. 2.—*A.* Cochlear nerve function in ideal case for operation. *B.* Cochlear nerve function in borderline case for operation.

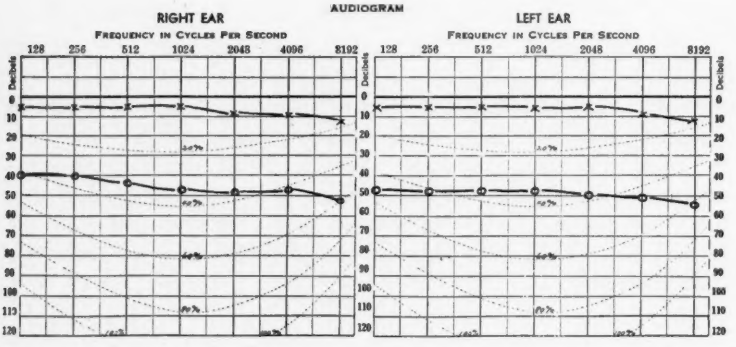
AUDIOGRAM
RIGHT EAR
FREQUENCY IN CYCLES PER SECOND
LEFT EAR
FREQUENCY IN CYCLES PER SECOND
128 256 512 1024 2048 4096 8192
Decibels
0 10 20 30 40 50 60 70 80 90 100 110 120

Fig. 3.—Audiogram of an ideal case for the fenestration operation with essentially equal hearing loss in both ears. Note the cochlear nerve loss is not greater than ten decibels in the speech frequencies.

In this and the following figures bone conduction is represented by x—x—x; air conduction by o—o—o.

Patients over 60 years of age should rarely be operated on. If a patient has good nerve function at this age, he will hear well with a hearing aid for the remainder of his life. Therefore, why subject this individual to the risks of a major surgical procedure?

How is cochlear nerve function determined?

Unfortunately, there is no exact method of determining cochlear nerve function. However, until better methods are developed, repeated audiometric tests, with a frequently calibrated bone oscillator, together with the data obtained from a 1024 magnesium alloy tuning fork, have proven to be reasonably accurate. Our patients are tested in a partially soundproof room and the bone oscillator is frequently calibrated and adjusted by four normals in the office. An audiologist also tests each patient independently in a suspended soundproof room, and we have found the preoperative and postoperative testing results in the two offices to be quite consistent.

Patients are classed as ideal, borderline, or non-suitable for the fenestration operation (Fig. 2).

1. In the ideal case the cochlear nerve loss must not exceed 10 decibels in the speech frequencies.

2. In the borderline case the cochlear nerve loss must not exceed 20 decibels in the 512 and 1024 frequencies, and must not be greater than 30 decibels at the 2048 level.

3. Any patient having a cochlear nerve loss below the borderline group is generally considered to be non-suitable for surgery.

Occasionally one may be justified in operating on a so-called non-suitable case. Especially is this true in young individuals who have had a rapid loss of both air and cochlear nerve function occur in a brief period of time. This type of case often rapidly progresses to advanced deafness. Therefore, surgery may be occasionally justified in these cases in an attempt to prevent further nerve deterioration.

I might digress here for a moment to say the exact cause of cochlear nerve deterioration in otosclerosis is not known. However, surgery with successful restoration of hearing to the serviceable level seems to inhibit further deterioration of cochlear nerve function. Obviously, time alone will answer this pertinent question.

Any patient with symptoms of conduction deafness who hears well with a hearing aid may be suitable for operation. If this patient hears a 1024 tuning fork longer by bone than by air conduction he is, in all probability, a suitable case. If, on the other hand, the patient

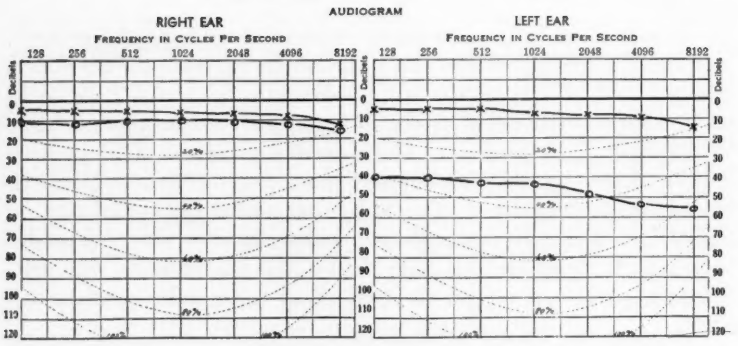

Fig. 4.—Audiogram of patient having unilateral otosclerosis.

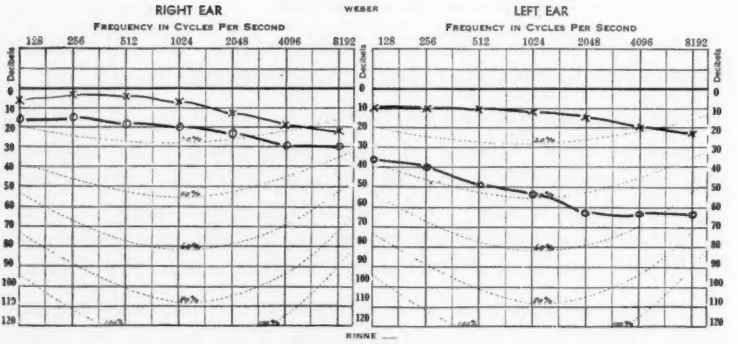

Fig. 5.—Audiograms showing bilateral otosclerosis, with serviceable hearing in the right ear.

hears a 1024 tuning fork longer by air than by bone conduction, he can almost be eliminated as a candidate for operation. Subsequent audiometric studies will usually substantiate these tuning fork findings.

Assuming the patient has lost serviceable hearing, the degree of air conduction loss is immaterial in the selection of cases for operation as long as the cochlear nerve function is reasonably good (Fig. 3).

All other factors being equal, the poorer of the two ears is usually selected for operation, although the patient's choice is very important in this matter. If the patient has experimentally found the hearing aid is more effective in the poorer ear, or if the tinnitus is less noticeable in the poorer ear, then it may be advisable to operate on the better ear. If hearing studies reveal both ears to be essentially the same, the choice of the side to be operated on is determined by the patient (Fig. 4).

With essentially normal hearing in one ear, are we ever justified in operating on the opposite otosclerotic ear?

This procedure is justified only on very rare occasions. If tinnitus is unbearable in the otosclerotic ear, it may at times be alleviated by an operation. Occasionally certain occupations, such as railroading, demand binaural hearing and on the basis of economic necessity, an operation on the otosclerotic ear may be justified (Fig. 4).

If one has evidence of bilateral involvement, but the hearing in one ear is still serviceable, should the poorer ear be operated on?

Ordinarily we do not advise surgery unless the patient has lost serviceable hearing in both ears. However, these patients should be carefully observed and if there is evidence of progressive nerve loss in the poorer ear then surgery should be done on this ear in spite of the serviceable level in the better ear. This is carried out in an attempt to prevent further cochlear nerve deterioration and to operate on the poorer ear before it becomes non-suitable for fenestration (Fig. 5).

Is surgery indicated during pregnancy?

If the patient with otosclerosis gives a history of hearing loss during a previous pregnancy, a fenestration operation may be done early in a subsequent pregnancy in an attempt to prevent further hearing loss. We have operated on six such patients during the third or fourth month of pregnancy without encountering any undue difficulty.

If the patient has had no previous pregnancies or gives a history of previous pregnancies without hearing damage, then only observation is indicated, as many patients will go to term without showing evidence of further hearing loss.

If the pregnant patient with otosclerosis exhibits a marked cochlear nerve loss and is, at the time of examination, non-suitable for fenestration operation, I would be inclined to advise termination of the pregnancy, especially if the patient gives a past history of increased hearing loss during a previous pregnancy. The question of therapeutic abortion in otosclerosis has many problems which demand careful scrutiny. The psychological effect of this procedure is tremendous and consultation not only with the obstetrician, but with another otologist, is always advisable. The ultimate decision, of course, must rest with the patient.

Complications of Fenestration Surgery.—Having determined the suitability of the patient for fenestration, we now answer the patient's second question: "What are the possible complications of such a procedure?"

Every patient contemplating a fenestration operation should be fully aware of the possible complications that may develop. Fortunately, in this series of 500 cases, complications of a serious nature seem to be relatively negligible. There were no mortalities and no serious operative complications, such as facial nerve injury, lacerated membranous labyrinths, lateral sinus or dural perforations.

Seven patients, representing 1.5 per cent, developed a temporary facial paralysis and subsequently completely recovered. This complication developed one week to ten days following surgery and subsided within one month after the onset.

An extremely annoying, but not incapacitating vertigo continues to be present in six patients, representing 1+ per cent of those operated on. Postoperative vertigo is most marked the first two or three days after surgery and usually completely subsides during the first four to six weeks following surgery. Some patients, however, continue to be troubled on sudden motion of the head for an indefinite period of time.

Eight patients developed otitis media following surgery, which resulted in a permanent opening in the ear drum in four cases. Perforation of the ear drum occurred during surgery in nine cases, apparently at the time the neck of the malleus was sectioned. Five

of these drums subsequently healed and the remaining four failed to close. A perforation of the ear drum definitely impairs the hearing result.

Aural discharge, usually nocturnal and of a mucoid character, is bothersome in some 20 per cent of the cases. This drainage may be intermittent or continuous and at times becomes secondarily infected. Routine cultures of ears draining longer than two months reveal Staphylococcus aureus to be the most common organism, although Bacillus fecalis has been isolated in two instances. It is my belief this discharge is usually the result of incomplete epithelization of the cavity. In these cases the removal of small areas of granulation tissue by means of a small curette is often necessary before epithelization is finally completed. Since using the single incision and creating a smaller mastoid cavity, complete epithelization has occurred more rapidly and the incidence of postoperative drainage has been materially reduced.

Results Obtained By Fenestration.—Having determined the patient's suitability for surgery and explained the possible complications, we now answer the third question: "What results may I expect following the fenestration operation?"

Four hundred and twenty-four, or 85 per cent, of the 500 patients in this series complained of preoperative tinnitus. The following chart reveals 24 per cent of these were completely relieved and an additional 54 per cent were partially relieved of this complaint. Therefore, a total of 78 per cent of the patients operated on noted a definite improvement in their tinnitus. It is interesting to note the group that obtained the greatest benefit also gained serviceable hearing following surgery.

TABLE. 1.—EFFECT OF FENESTRATION ON TINNITUS

TINNITIS	RELIEVED	DECREASED	UNCHANGED	INCREASED
Serviceable Hearing	80 or 19%	168 or 42%	21 or 5%	5 or 1%
Non-serviceable Hearing	21 or 5%	61 or 12%	52 or 13%	13 or 3%
Total Cases	101 or 24%	229 or 54%	73 or 18%	18 or 4%

Hearing results are evaluated by correlating the findings of our questionnaire (Fig. 6) with our audiometric studies at six months, one year, and two years postoperatively.

Dear

It has been_____months since your hearing operation. Will you please complete the following questionnaire and return it promptly for my records?

Question	Yes	No
1. Do you have practical useful hearing?	☐	☐
2. Is your hearing improved?	☐	☐
3. Is your hearing unchanged?	☐	☐
4. Is your hearing worse?	☐	☐
5. Did you wear an aid before surgery?	☐	☐
6. Do you wear an aid now?	☐	☐
7. Do you feel the need of an aid?	☐	☐
8. Did you have head noises before surgery?	☐	☐
9. Are your head noises now (gone ☐; less ☐; worse ☐)?		
10. Do you have ear discharge?	No ☐ Slight ☐ Marked ☐	
11. Do you have dizziness?	No ☐ Slight ☐ Marked ☐	

Any comments by relatives or friends regarding your hearing will be appreciated.

Fig. 6.—Questionnaire.

We are now testing patients by speech audiometry. This may explain on the basis of speech intelligibility the apparent discrepancy which sometimes occurs in comparing the patient's questionnaire with the audiometric findings In most cases, however, the patient's questionnaire coincides with the 30 decibel level established for serviceable hearing.

TABLE 2.—HEARING RESULTS BASED UPON CLASSIFICATION.

266	Ideal Cases	213	or 79%	Serviceable Hearing
211	Borderline Cases	137	or 66%	Serviceable Hearing
23	Non-suitable Cases	1	or 4%	Serviceable Hearing
500	Total Cases	351	or 70%	Serviceable Hearing

It is interesting to note there is a 13 per cent difference in prognosis between the ideal and the borderline case. The chances of restoring serviceable hearing in the so-called non-suitable patient are very poor indeed.

TABLE 3.—HEARING RESULTS OF ALL OPERATED CASES.

Serviceable hearing	351 or 70%+
Improved hearing but non-serviceable	86 or 17%
Unchanged	44 or 9%
Closure of fenestra	39 or 8%
Air conduction worse than preoperatively	19 or 4%
Bone conduction worse than preoperatively	5 or 1%

In this series of 500 patients, 39, or 8 percent, lost their initial hearing gain due to closure of the fenestra within the first six months following surgery. An additional 3 per cent closed during the second six months. To date we have not had a closure occur in any patient after the first year. Therefore, after one year 66+ per cent of all patients operated on in this series have maintained serviceable hearing.

Closure of the fenestra has occurred in any patient who obtained improved hearing and subsequently permanently lost all or any part of the original gain, providing, of course, there is no other obvious ear pathology to account for this hearing loss. A positive fistula test may or may not be present in these cases, depending on whether the closure is partial or complete and whether it is due to fibrous tissue or to bony growth.

TABLE 4.—CLOSURE OF FENESTRA.

TIME POST-OPERATIVE	NO. OF CASES	CLOSED FENESTRAE
1 to 6 Mo.	500	39 or 8%
6 to 12 Mo.	275	12 or 4%
12 to 24 Mo.	40	0 or 0%
Total closed fenestrae		51 or 12%

If closure of the fenestra develops, it usually occurs in the third or fourth month following surgery. If the patient has serviceable hearing at six months, he apparently has a 97 per cent chance of permanently maintaining his hearing gain, as far as closure is concerned.

When closure occurs in one ear, it does not indicate that closure will also occur in the opposite ear if it is operated on.

The second ear should not be operated on if the hearing did not reach the serviceable level in the first ear unless the patient is able to hear well with a hearing aid in the originally operated ear. Occasionally, speech reception through an aid is very distorted in a fenestrated ear, even though no apparent change has occurred in the cochlear nerve function. It is my opinion this may be due to the effect of the enlarged cavity on the reception of sound.

It has been my custom to keep a notebook in the operating room in which all details of the individual operation are jotted down immediately following the operation. Included are notations concerning the bleeding, stapes fixation, dural exposure, thinness and size of the flap, and position and size of the fenestra, as well as notes regarding any minute endosteal remnants or bone dust that may remain in the fenestra. Subsequent correlation of this data is most interesting and instructive.

TABLE 5.—FENESTRA DETAILS IN 500 CASES AT SIX MONTHS.

	TOTAL CASES	WITHOUT REMNANTS	ENDOSTEAL REMNANTS	BONE DUST	BOTH ENDOSTEUM AND BONE PARTICLES
Patent Fenestrae	461 or 92%	308 or 67%	60 or 13%	93 or 20%	41 or 9%
Closed Fenestrae	39 or 8%	27 or 69%	5 or 13%	7 or 17%	4 or 10%

It appears from the above figures that the presence or absence of minute remnants of endosteum or bone dust in the fenestra is not the only cause of closure. It is interesting to note that one-third of the open fenestrae, as well as one-third of the closed fenestrae, had some very minute remnants of endosteum or bone dust remaining in the fenestra at the time surgery was completed. I must stress, however, in spite of this finding every effort must always be made to remove completely each particle of endosteum or bone dust from the fenestra in every case. Since we have been using the double blue line technique and elevating the intact endosteum in most cases the incidence of remnants in the fenestra has been materially reduced.

Revision surgery is indicated in any patient who obtained serviceable hearing which subsequently returned to the pre-operative level, providing the cochlear nerve function has remained stationary.

If serviceable hearing was not obtained after the initial operation, a revision should not be done.

In general, the longer one waits before performing a revision, the better the prognosis. I prefer to wait one year after closure has occurred, hoping the osteogenic process is no longer active. If a bony plate is found over the fenestra which can be elevated and removed without the use of a burr, closure at a later date is less likely to occur. Great care should be exercised in raising the flap in revisions, for if fibrous tissue has adhered to the membranous labyrinth, damage to the inner ear may occur.

TABLE 6.—REVISION RESULTS.

CASES	NON-SERVICEABLE HEARING	SERVICEABLE HEARING
16	12 or 75%	4 or 25%

Twenty-five per cent of our patients who had revisions have maintained serviceable hearing longer than six months.

CONCLUSIONS

The fenestration operation is not a cure-all for otosclerosis. There are many causes of failure which remain to be solved and which serve as a challenge to those of us interested in this field of endeavor.

I would like to again stress the importance of thoroughly acquainting the patient with otosclerosis with all the facts of fenestration surgery.

1. The importance of cochlear nerve function cannot be overemphasized in determining a patient's suitability for fenestration surgery.

2. Serious complications in fenestration surgery seem to be relatively negligible.

3. In this series of 500 patients, approximately 66 per cent have maintained practical hearing after one year.

1136 WEST SIXTH STREET.

REFERENCES

1. Lempert, J.: A New Oval Window for the Improvement of Hearing in Cases of Otosclerosis, Arch. Otolaryng. 34:880 (Nov.) 1941.

2. Lempert, J.: Lempert Fenestra Nov-Ovalis With Mobile Stopple, Arch. Otolaryng. 41:1-41 (Jan.) 1945.

3. Meals, Wm. G.: Anesthesia in Fenestration Surgery. Paper read before the California State Medical Meeting, 1946.

4. McNaught, Robert: Personal communication.

5. Shambaugh, G. E., Jr.: A Modified Fenestration Technic, Arch. Otolaryng. 36:23 (July) 1942.

6. Shambaugh, G. E,. Jr.,: Surgical Treatment of Otosclerosis, Arch. Otolaryng. 43:549 (June) 1946.

IV

REEVALUATION OF THE IMPLANTATION OF FASCIAL STRIPS THROUGH THE MASSETER MUSCLE FOR SURGICAL CORRECTION OF FACIAL PARALYSIS

(REPORT OF THREE ADDITIONAL CASES)

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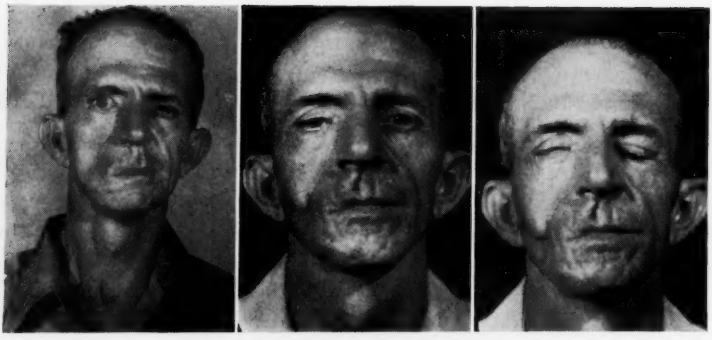
NEW ORLEANS, LA.

In November 1946, we presented our technique and results in fascial and muscle transplants for the correction of facial paralysis. Since our original presentation we have had the opportunity to observe these patients further and to operate on three new patients and reoperate on two of our previous series.

At this time there is no need to reiterate the various techniques and theories that have been presented in the past by surgeons employing these procedures. Suffice to recall that Lexer in 1908 was the first to employ a muscle transplant for this purpose and in 1913 Busche used fascia lata. The masseter muscle and in some instances the temporal muscle have been utilized either directly or by their attachment to a fascial sling to support the obicularis oris. Most commonly the fascial strips have been attached to the temporal muscle to give support for the obicularis oculi. Utilization of the fascia of the temporal and associated muscle bundles has also been employed. We have used the masseter muscle both for support to the obicularis oris and the obicularis oculi.

We are of the opinion that the direct attachment of the muscle fasciculi to the paralyzed muscles offers a less satisfactory result than the use of fascial strips which connect the masseter muscle to the paralyzed one. The latter procedure does not risk possible transection of the nerves to the masseter nor does it impair its forcefulness by severing its normal attachment. Furthermore, there is less danger of loss of muscle tissue due to the secondary complication of infection. Unquestionably, in some of the cases considerable fibrosis of the muscle occurs, thus impairing its activity.

Read before the Southern Section of the American Laryngological, Rhinological and Otological Society, New Orleans, La., Jan. 23, 1948.



A *B* *C*

Fig. 1, Case 1.—*A.* Photograph of a 42-year-old white male with facial paralysis involving the right seventh nerve. The patient had a fascial implant on August 13, 1946, with subsequent infection with some drainage which caused a slight separation of the wound sufficient to reveal a portion of implanted fascia. Because of apparent swelling at this site, an interne incised the wound and excised all visible fascia at this point. On July 13, 1947, another fascial implant was made along with subzygomatic fascial transplant from the masseter to the obicularis oculi. Despite the fact that no oral penetration was made, a second infection developed which showed considerable discharge of gelatinous mucoid substance. This infection subsided and drainage ceased. Subsequent to wound healing, the patient had good function.

B. Photograph showing patient in repose following second operation. Note there is still some edema at the site where drainage occurred but though drainage is still not checked, the right angle of the mouth is brought up in a position which shows considerable improvement. Note also that the palpebral opening has been narrowed as a result of the subzygomatic implant from the masseter to the obicularis oculi muscles.

C. Photograph showing patient in repose with lids closed. Note ability of patient to approximate lids in much more normal fashion.

In one instance, immediately upon severing the masseter muscle from its inferior attachment and before splitting the fasciculi, an unexplainable loss of tone was apparent. In a previous case in which the muscle fasciculi were used the resulting correction was insufficient and necessitated a fascial sling type of operation. Occasionally, the direct muscle anastomosis is indicated because it serves an additional purpose of filling a defect of contour.

The utilization of fascial strips attached directly to an intact muscle, which has normal innervation, and carried to the paralyzed muscles furnishes a supportive sling that gives an additional quality

of movement upon voluntary contraction of the normal muscle. The masseter muscle, innervated by the trigeminal nerve and firmly attached to the zygomatic arch by a strong tendinous attachment and to almost the entire ramus of the mandible, is particularly well suited for this purpose. The slight obliquity of attachment and the vertical contraction of the muscle provide a suitable directional pull on the affected muscle.

The operation is performed under local infiltration anesthesia. As previously, we utilize fascia lata obtained from the thigh either by use of a vein stripper or by direct excision. We feel that the submandibular incision is to be considerably preferred to an incision over the masseter muscle because of the better cosmetic result obtained from this incision and also because of the opportunity afforded for facile extension of the incision anteriorly to the ear for increased exposure or the utilization of the temporal muscle. If the additional face-lift type of procedure to remove excess skin is desirable, no additional scarring will result.

Anterior fasciculi of the masseter muscle are isolated and tested for power of contraction by asking the patient to bite down. A section of suitable size, about one-third to one-half of the muscle bundle, is selected for use. Then a Blair fascia carrier is inserted and carried through the subcutaneous substance of the cheek and lip to a point past the midline of the upper lip. A skin incision is made and the fascia strip is then grasped and pulled into position along the tract as the Blair carrier is withdrawn. The same procedure is again carried out in a path parallel to the first tract. The fascia carrier is inserted, carried subcutaneously in a path beneath the vermilion cutaneous juncture to the point past the midline where the skin incision was made. The other end of the fascia after encircling a portion of the obicularis oris muscle is carried back to the masseter area as the Blair carrier is withdrawn. A similar procedure is carried out on the lower lip and a fascial strip coursing from the masseter to a point past the midline and back to the masseter area is established. One-half of the fascial loop is then threaded beneath the selected masseter fasciculi and after the fascia is drawn taut, it is knotted and a second knot tied. These are then transfixed with rayon sutures. The extent of overcorrection, that is, the tautness of the fascial loops, is difficult to estimate but such tension as will draw the mouth backward approximately 2.5 cm. is about correct. After establishing the fascial loops to the lower lip those of the upper lip are laced beneath the muscle, drawn taut, knotted and fixed in an identical manner.

A B

C D E

Fig. 2, Case 2.—*A.* Photograph of 32-year-old female with left-sided facial paralysis, resulting from infantile paralysis which developed at the age of four. Note the typical stare of the left eye and the drooping of the left angle of the mouth.

B. Photograph showing immobility of the paralyzed muscle on the left when patient attempts to effect backward pull of the obicularis oris muscle. These patients frequently show exaggerated muscle pull on the unaffected side.

C. Photograph showing this patient with face muscles in repose following implant of fascial strip to the masseter muscle on the left side. Note the angle of the mouth is at the normal level. Note also narrowing of the left palpebral fissure.

D. Photograph showing patient with facial muscles in repose with the angle of the mouth in the same normal position. Note the patient's ability to close the eyelids almost completely on the paralyzed side following fascial transplant to the left upper eyelid.

E. Photograph showing amount of animation which is possible from the pull of the masseter muscle on the implanted fascial strips following operation for attempted correction of the paralysis on the left side.

In fixing the fascia to the masseter muscle it should be fixed so that it is attached to a point superior to the normal angle of the mouth to assure an upward pull. Care not to enter the oral cavity is extremely important for contamination and secondary infection negate the end result.

In attaching the fascia to the obicularis oculi muscle, we have, on two occasions, carried the fascia beneath the zygoma to a point just lateral to the outer canthus. There it is brought out through the skin and one-half carried beneath the skin through the upper eyelid and the other through the lower lid to the inner canthus where it is again brought out through a skin incision, tied to its mate, which has coursed through the opposite lid, and is sutured with nylon and tacked to the periosteum of the nasion, adjacent to the region of the inner canthus.

The skin incisions are then closed with subcutaneous sutures of nylon and the skin with dermalon 00000. Strips of one-inch gauze are fixed by collodion to the skin for support. These strips are first attached to the lip area and then carried backward and upward in a path approximating that of the fascia and attached superiorly to the skin over the zygomatic area for support. In applying such strips to the upper lip care should be taken not to cross cut the ala of the nose because postoperative swelling may cause a pressure necrosis.

Postoperatively, these supportive strips are reapplied as frequently as necessary to maintain adequate support for a period of at least one month. Because of support furnished by this type of dressing the sutures may be removed on the third postoperative day. The patient is on a liquid and semi-soft diet for the first month. Thereafter, he is permitted to contract the muscles and later instructed to practice coordinated facial movements by practicing before a mirror.

We feel that when three years have elapsed since the transection of the nerve; when direct nerve anastomosis is not feasible; when the substitution of the spinal accessory or the hypoglossal nerve by anastomosis is not possible or is undesirable because of the resultant paralysis of the shoulder or tongue muscles; or when a nerve anastomosis has been attempted previously and failed; then this procedure offers much to those afflicted with facial paralysis regardless of the etiology.

In our series the facial paralysis has been due to surgical transection in excision of a tumor, the excision of a neuroma acoustica,

A *B*

C *D* *E*

Fig. 3, Case 3.—*A.* Photograph of patient showing tumor of the parotid which is just posterior to the angle of the left jaw. It was necessary to extirpate the entire tumor of the parotid gland within its capsule, which naturally resulted in complete facial paralysis on left side.

B. Photograph following extirpation of the parotid gland which resulted in left-sided facial paralysis. There is little drooping of the left angle of the mouth despite the fact that the entire seventh nerve was removed but the patient shows typical stare in the left eye.

C. Photograph showing patient following operation with muscles in repose. Note that the left angle of the mouth is in fairly normal position. No operation for correction of the paralysis of the lid has been done at this stage.

D. Photograph showing amount of animation resulting from operation which enabled the patient to purse the lips.

E. Photograph reveals further animation that has resulted from operation and patient can coordinate movement in moderate degree of smiling.

residual permanent paralysis following Bell's palsy, infantile paralysis, and causes unknown.

If fascial strips are utilized, there is little cause to destroy the innervation of the normal muscle and unless the result is impaired by a secondary complication, it does offer more than static support to the involved side of the face.

In our previous discussion of this technique we reported 11 cases. At this time we wish to correct our statement in regard to the report of cases. At that time we had performed a total of 11 operations on eight patients. At this time we wish to present five additional operations, two of which were on patients previously operated on and three new ones.

REPORT OF CASES

CASE 1.—DeT. This patient, aged 42, had a facial paralysis. On August 13, 1946, he was first operated on by us, at which time through a submandibular incision a fascia transplant to the obicularis oris muscle was made. Due to secondary infection the result was impaired. The fascia appeared at the point of drainage and an uninformed interne forcibly assisted its exitus. On July 13, 1947, through a lateral incision directly over the masseter muscle a fascia transplantation to the obicularis oris and the obicularis oculi was accomplished. The fascia again was subzygomatic. Despite the fact that there was no oral puncture the wound became grossly infected and the discharge which was late in occurrence was gelatinous and of a suggestive mucoid character. Ultimately the infection subsided, the drainage ceased, and at the present time he has good function.

CASE 2.—B. W. This 32-year-old patient suffered from infantile paralysis at the age of four with residual dysfunction of the facial nerve on the left side. She was operated on by us on June 7, 1945, November 26, 1945, and August 5, 1946. On June 23, 1947, we used a muscle fascia transplant to the left eye and a fascia transplant to the upper lip. Functionally, the results are satisfactory but the cosmetic result leaves much to be desired owing in part to the placement of the cheek incision.

CASE 3.—E. This 31-year-old patient had a mixed tumor of the parotid excised on May 20, 1947. It was necessary to transect the facial nerve and there was a residual depression in the parotid gland area. On November 4, 1947, a muscle transplantation to the upper and lower lip was performed. At the time of operation although the transplantation was technically satisfactory, immediately

A B

C D

Fig. 4, Case 4.—*A.* Photograph of 30-year-old patient who gradually developed facial paralysis associated with left vocal cord paralysis due to an unknown cause. The loss of function occurred during the period of 1943 and 1944. This patient has previously had an anastomosis between the seventh and ninth nerves but at the time of initial examination there was no evidence of innervation in the distribution of the seventh nerve.

B. Photograph of patient with face in repose. Notice the slight drooping of the left angle of the mouth with typical drooping involving the left eyelids. The droop of the mouth is less pronounced in this case than is usually seen.

C. Photograph of patient following operation for fascial transplant attached to the masseter muscle and the paralyzed muscle about the angle of the mouth, which was done November 11, 1947. No attempt was made to correct the paralysis of the eyelid at this time. Note the raised angle of mouth on left which resulted from the fascial implant. This is revealed by this photograph of the patient in repose.

D. Photograph of same patient in repose showing inability to close the eyelids completely on the paralyzed side.

upon transection of the muscle from the mandible there was an apparent loss of activity. At the present time she has not presented the degree of motion characteristic of a fascial transplant at a comparable postoperative period. The case does illustrate the usefulness of the procedure with regard to giving a better contour in the case of a surgical concavity. Certainly, it is to be preferred to a dermal or cartilaginous transplant which would confuse the surgical field or future operations to obtain movement.

CASE 4.—S. This 30-year-old patient had a gradual facial paralysis with left vocal cord paralysis due to an unknown cause. The loss of function occurred during the two-year period of 1943-44. Twenty-one months prior to being seen by us she had an anastomosis between the seventh and ninth nerves and the neurosurgeons stated that there was some improvement. At the time of examination by us there was no evidence of innervation in the area of distribution of the seventh nerve. On November 11, 1947, a fascia transplant from the masseter muscle to the upper and lower lips was made and it was satisfactory both from the technical and the functional point of view. She has good motion in the immediate postoperative period and a transplantation to the obicularis oculi is contemplated.

CASE 5.—S. This 27-year-old female suffered a facial paralysis at the age of nine from an unknown cause. On February 4, 1947, through a submandibular incision, a fascial transplantation from the masseter to the obicularis oris muscle was performed. There were no postoperative complications and function was satisfactory. Through the same submandibular incision the masseter muscle again was exposed on July 22, 1947. The transplantation to the obicularis oris was repeated and a fascial transplantation through the subzygomatic route to the obicularis oculi was performed. At the present time the patient has motion and her only complaint is an inability to open the involved eye as wide as the unaffected one.

1615 AMERICAN BANK BUILDING.

REFERENCES

1. Adson, A. W.: Surgical Treatment of Facial Paralysis, Arch. Otolaryng. 2:217-249 (Sept.) 1925.

2. Alexander, R. J.: Correction of Facial Paralysis by Muscle Transplant, Rocky Mt. M. J. 38:713-716 (Sept.) 1941.

3. Blair, V. P.: Further Observation upon the Compensatory Use of Live Tendon Strips for Facial Paralysis, Ann. Surg. 92:694-703 (Oct.) 1930.

4. Blair, V. P.: Notes on the Operative Correction of Facial Palsy, South. M. J. 19:116-120 (Feb.) 1926.

5. Brooke, R.: A Case of Facial Paralysis Treated by Fascial Grafts, Brit. J. Surg. 20:523-526 (Jan.) 1933.

6. Brown. J. B.: The Utilization of the Temporal Muscle and Fascia in Facial Paralysis, Ann. Surg. 109:1016-1023 (June) 1939.

7. Brunner, H.: Plastic Operations in Paralysis of the Facial Nerve, Arch. f. klin. Chir. 140:85-100, 1926.

8. Bunnell, S.: Surgical Repair of the Facial Nerve, Arch. Otolaryng. 25:235-259 (Mar.) 1937.

9. Busch, H.: Cosmetic Improvement of the Deformity Produced by Facial Paralysis, Ztschr. f. Ohrenh. 68:175, 1913.

10. Busch, H.: The Cosmetic Treatment of Facial Nerve Paralysis, Beitrage z. Anat. Physiol. Path. and Ther. d. Nase u. d. Hales 3:380, 1910.

11. Fenwick, G.: Surgical Treatment of Facial Paralysis, Brit. M. J. 2:700 (Nov.) 1919.

12. Fischer, H.: Traumatic Facial Paralysis and Its Surgical Treatment by Free Transplantation of Fascia Lata, Ann. Surg. 89:334-339, 1929.

13. Halle, Max: The Occurrence of Facial Paralysis and Methods for Its Correction, Laryngoscope 48:225-235 (April) 1938.

14. Hanrahan, E. M., and Dandy, W. E.: A Procedure to Correct Facial Paralysis, J. A. M. A. 124:1051-1054 (April) 1944.

15. Hastings, S.: Transplantation of Anterior Half of Masseter Muscle for Facial Paralysis, Proc. Roy. Soc. Med., Lond. (Sect. Otol.) 13:64, 1919-1920.

16. Jianu, Amza: The Surgical Treatment of Facial Nerve Paralysis, Deutsche Ztschr. f. Chir. 102:377, 1909.

17. Lathrop, F. D.: The Repair of Traumatic Lesions of the Facial Nerve Secondary to War Wounds, S. Clin. North America, pp. 763-774, June 1946.

18. Lodge, W. O.: A Plastic Operation for Facial Paralysis, Brit. J. Surg. 17:422 (Jan.) 1930.

19. Morestin, H.: Section of the Factal, Lingual and Superior Maxillary Nerves by the Same Projectile. Tentative Improvement of the Facial Paralysis by Muscular Anastomoses, Bull. et mém. Soc. d. Chir. de Paris, 41:1370, 1915.

20. Owens, Neal: Implantation of Fascial Strips Through the Masseter Muscle for Surgical Correction of Facial Paralysis; A Report of 11 Cases, Plastic and Reconstructive Surg. 2:25-36, 1947.

21. Perthes, G.: Is Nerve Grafting or Plastic Muscle Operation to be Preferred in the Treatment of Irreparable Facial Nerve Paralysis? Zentralbl. f. Chir. 51:2073, 1924.

22. Pickerill, P.: Facial Paralysis, Palatal Repair and Some Other Plastic Operations, M. J. Australia 1:543-548 (May) 1928.

23. Schmid, B.: The Operative Treatment of Irreparable Facial Nerve Paralysis, Zentralbl. f. Chir. 65:1296 (June) 1938.

24. Stein, A. E.: Cosmetic Correction of Facial Nerve Paralysis by Means of Free Fascia Nerve Plastic Operation, München. med. Wchnschr. 60:1370, 1913.

V

SPECIAL TECHNIQUES FOR THE DIAGNOSIS AND TREATMENT OF PSYCHOGENIC DEAFNESS*

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An increasing awareness of psychogenic deafness as a clinical entity has developed within the past few years. Because of the obvious difficulties in the diagnosis and treatment of psychogenic deafness, there is no clear understanding of the respective responsibilities of the otologist and the psychiatrist in handling the individual case, nor of the means with which cooperative handling may be undertaken. The present discussion is designed to point up some of the clinical problems, to outline some simple techniques that have proved serviceable in diagnosis and treatment, and to submit a group of selected case reports in illustration of these techniques.

The incidence of psychogenic deafness is not known. Reports from the Army and Navy centers suggest an incidence of from ten to twenty per cent among personnel on active duty who were hospitalized for the treatment of severe hearing disability.[5, 10, 11] How much the military environment contributed to this finding is difficult to evaluate, and it is even more difficult to correlate the military and the civilian situations. Informal discussions with several persons concerned with psychogenic deafness in the military centers indicate that in many instances the etiologic picture has shown no direct relation to a military environment, that the problems involved were such as might be encountered in daily living. That psychogenic deafness exists as a recognizable entity, that it may be encountered fairly frequently, and that its apprehension and treatment require far more attention than is commonly given, are realities that seem to lie beyond further argument.[7, 8]

The clinician's first concern is with accurate diagnosis. Assuming that psychogenic deafness is a psychosomatic disorder, the clin-

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*The term "psychogenic deafness" is, of course, merely the brief and accepted way of referring to the psychic element in the impairment of hearing manifested by some persons.

ician must be able to differentiate between the psychologic accompaniments of organic deafness and a psychogenic involvement that operates causally in such a way that some maladjustment of the personality is referred to the ear itself.[11, 12] Much of the clinical work reported to date, particularly that involving the use of intravenous injections of one of the barbiturates, has been such that the diagnosis is not clearly established until therapy has been accomplished.[5, 10] Granted that some excellent results have been achieved in military medicine by the use of a barbiturate as a means of bringing subconscious conflict to the surface, "diagnostic therapy" (or "therapeutic diagnosis") remains a clinical anomaly. Moreover—and contrary to some commonly held beliefs—the failure to evoke the emotional conflict or to demonstrate improved hearing in a single session with intravenous barbiturate injection is by no means uncommon and does not necessarily confirm organic deafness.

No single factor in the clinical findings is clearly indicative of a psychogenic involvement in hearing disability. Stress has been given to the inconsistency of responses to routine retests as a sign of psychogenic involvement, and to the failure of test-responses to fit the classical findings of otic pathology. There is no typical pattern in this inconsistency. On occasion, it is necessary for the clinician to "throw the book" of tests and retests at the patient, and even then there may remain some doubt. The conviction has grown, therefore, that one cannot be absolutely certain he is dealing with psychogenic deafness until he has effected a cure.

The evidence to date indicates that a psychogenic hearing involvement may range in causal terms from malingering (as a symptom of maladjusted behavior) to hysteria (wherein the conversion symptom is an involuntary expression of emotional conflict at the subconscious level).[3, 8, 10] Hysterical deafness of classical description seems to occur relatively infrequently. The psychogenic overlay on organic disease is by all means the most common type of psychogenic hearing involvement.[3, 8, 10, 11, 12]

Accordingly, the task of diagnosis is not only to differentiate psychogenic from organic deafness, but also to measure the extent of a psychogenic involvement that frequently accompanies or is in some way related to otic lesions. This puts the problem of diagnosis squarely up to the otologic clinician, for his special knowledge of the pathology and physiology of the hearing mechanism must be brought to bear on a wide variety of clinical data. He is responsible for determining the consistency of test-findings and the deviation from classical patterns of otic pathology.

Once the diagnosis has been made and the possible relation between organic and psychogenic dysfunction has been estimated, the otologist may wish to refer the patient for neuropsychiatric treatment and wash his hands of the problem. Often, however, because the patient believes his difficulty to be centered in the ear and because the symptom has developed as a release from behavioral conflict with unpleasant associations, he will resist psychiatric referral. He is resentful of an opinion that his is a "mental disorder"; he knows he is not crazy and says so. Yet, if the "ear doctor," the man who really knows about his trouble, can demonstrate to him that something different from or more than organic dysfunction is operative and can help him gain insight into the reasons for his symptoms, he will often respond quite readily. For obvious reasons, the therapeutic needs of each patient are unique.

In short, then, regardless of the ultimate disposition of the patient's problem, the otologic clinician must be prepared to make the diagnosis in terms as clear as possible. The primary need is the development of a reasonably simple technique to make this possible. Repeat tests are necessary. In order to refine the impressions gained from routine audiometry (both pure-tone and speech audiometry are necessary) and tuning-fork tests, the clinician may employ several special procedures. The Stenger and the Lombard tests are well known, and several modifications of them have been fully described.[3] More specific than these, perhaps, is the test recently devised by Doerfler and Stewart to isolate the malingerer and the "psychogenic."[2, 11, 12] Although this test can only suggest a possible ratio between psychogenic and organic involvements, it is reasonably specific in determining the presence of voluntary or involuntary repression of the hearing function.*

Routine and special tests thus furnish evidence of a disparate state of affairs. There yet remains the problem of the ratio between psychogenic and organic dysfunction and the need to determine the amount of psychogenic overlay. The use of suggestion hypnosis is recommended as a simple diagnostic procedure to fulfill this need. The otologist who depends upon suggestion hypnosis for diagnosis need not be dubious about its validity nor its effectiveness. As a

*The rationale of the Doerfler-Stewart test is the fact that the malingerer and the person with a psychogenic involvement seem usually to have adopted a subjective reference-level for their hearing. This fact was determined empirically. The use of superimposed signals of speech and masking noise, in measured amounts, disturbs this reference-level and, on several counts, the test points to deviations from the findings on normal and on pathologic ears. A similar use of masking noise as a nuisance-signal can also be effective with pure-tone audiometry.

diagnostician he is concerned with an accurate appraisal of the hearing function; he is not concerned with deep probing into emotional conflicts that may underlie the disorder nor with an attempt to bring subconscious maladjustments into consciousness. He simply wants to induce a state of control sufficient to relieve some of the inhibitions of the conscious state while he conducts routine tests of auditory acuity. This is made possible, readily enough, by suggestion hypnosis.

The technique is quite simple and can be mastered without formal instruction.[1] The "waking" technique is perhaps the simplest form, involving only fixation on a bright object to initiate the patient's response to deep suggestion. The result is a kind of hyperconcentration that induces complete relaxation very rapidly. Moreover, the "waking" technique is particularly useful with deafness, for a minimal demand is put upon the patient's speech-hearing function. Some verbal stimulus is called for, of course, and for this the use of a portable amplification system (microphone, small amplifier and headphones) is quite effective. Relatively few persons with a psychogenic overlay on organic disease are totally deaf for speech, and, even though a patient's hearing loss for pure-tone audiometry is profound, he can usually be reached by means of a powerful amplifier.

The clinician will have attained a useful depth of control over the patient when he can induce neuromuscular catalepsis by oral command. Quite often even a thoroughly relaxed state will prove sufficient for the purpose of auditory retest (and, incidentally, will often demonstrate the psychic component in many cases of viciously aggravated tinnitus). Some persons are resistive to the idea of hypnosis, both because of its association with mental disorder and because of exploitation of the hypnotic state in public entertainment. Others refuse to allow its use on religious grounds. For the most part, it is probably better to avoid the term "hypnosis" and talk instead of concentration and relaxation, and of the need to test the hearing while "the mind is free from tension and worry." Most patients are reasonably cooperative on this basis.

Once the patient is sufficiently "relaxed," the clinician conducts a brief audiometric retest, checking responses for both pure-tones and speech. If the test is extensive, it is necessary to reinforce the hypnotic state verbally; without reinforcement, light suggestion hypnosis lasts only a few minutes. Care must be taken to avoid false thresholds; it is possible for the patient to imagine that he hears a sound long after the intensity level is well below his threshold. Once it has been determined that the hearing threshold is better than it has proved to be under routine test circumstances, the clinician has

evidence—even if not quite exact—of the relationship between psychogenic and organic dysfunction. It is safe to assume that the thresholds so obtained indicate the maximum of organic impairment.

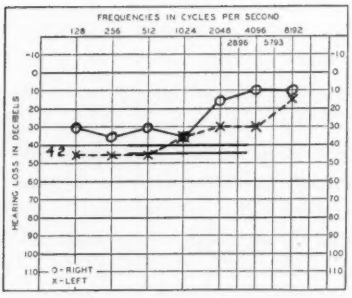
If a psychogenic overlay on organic disease is demonstrated by this technique, there is no harm in employing posthypnotic suggestion of improved hearing. Moreover, whatever the results, it is a good idea to suggest amnesia for the hypnotic experience, prior to awakening the patient.

Aside from the psychoneurotics whose symptoms relate to deep-seated fears, anxieties or conflicts and whose problems require extensive psychiatric analysis and treatment, there are many persons with a psychogenic hearing involvement who respond readily to simple psychotherapy. These are the persons whose problems are fairly close to the surface. The patient will often respond readily to the clinician's explanation of the referral of anxiety to the ear itself, a response that is best demonstrated by improved retest thresholds over a period of weeks. The essence of the therapy is the patient's ability to gain insight into his disability, to know why his maladjustment is associated with the hearing function. The success of the therapy is usually determined by the individual's willingness and ability to resolve his difficulties, once he realizes the connection between them and his hearing impairment.

REPORT OF CASES

Monaural-binaural testing was performed with headphones in matched calibration with the loudspeaker system. For the Lombard test, "white noise" from a custom-built noise generator was used; the Stenger test was done with the output of a standard audio-oscillator, amplified by a high-fidelity system and presented through matched, permanent-magnet headphones; the masking tone employed for the Doerfler-Stewart test was a square-wave at 128 c.p.s., with attenuation independent of that used for the speech signals.

In reference to the findings of the Lombard, the Stenger and the Doerfler-Stewart tests, the term "negative" means that the test gives no evidence of deviant behavior, while the term "positive" signifies some deviation from the recognized patterns of organic involvement.



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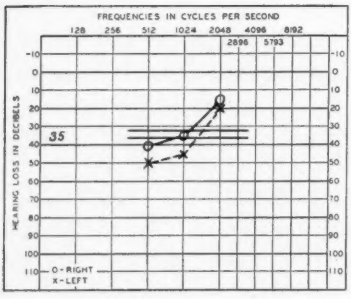

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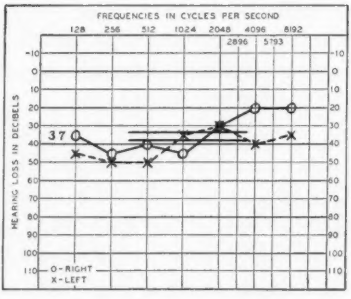


FINAL TEST

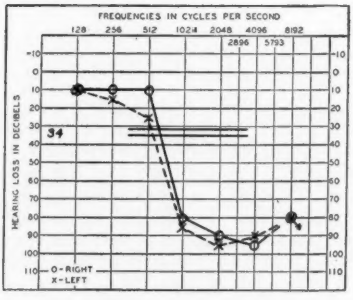
Fig. 1, Case 1.

NOTE.

The parallel lines on the audiograms indicate the threshold of speech-reception intelligibility in "free field" with the use of a loudspeaker in acoustically controlled conditions; the figures in the margin represent the speech-hearing threshold in decibels. The equipment used for speech-reception tests is a two-channel, high fidelity system calibrated to acoustic zero (.0002 dynes per square centimeter) at the test-point, capable of attenuation in one-decibel steps over a range of 100 decibels. All speech-reception testing was performed with the "live voice" technique; the test signals used were those in the spondee series, Harvard Psycho-Acoustic Laboratory Test No. 9.

CASE 1.—*CONTROL. Organic deafness, conductive type.* Age 19. Acute otitis media, 22 months previously, responded readily to chemotherapy; the hearing loss dated from that time. The tympanic membranes were of normal appearance; there was a light, bilateral, intermittent tinnitus. The bone conduction was prolonged and considerably better than air conduction in both ears; there was no lateralization from the midline. The patient gave a history of stuttering since early childhood, and of considerable facial distortion which had subsided within the previous four years. The speech pattern was fairly good; there was some retardation on initial stop-consonants, and an unpleasant nasal exhalation was employed as a releasing mechanism for the stutter-block.

Lombard and Doerfler-Stewart tests were negative. Because of the history of stuttering, it was felt that further diagnostic analysis might prove revealing. Under the influence of sodium pentothal, the hearing loss was further depressed. With the use of suggestion hypnosis the hearing thresholds were essentially the same as those obtained by routine testing. The 5-decibel shift in speech-reception threshold is explicable in terms of improved auditory habits. Therapy for the stuttering was undertaken during the course of aural rehabilitation (Fig. 1).



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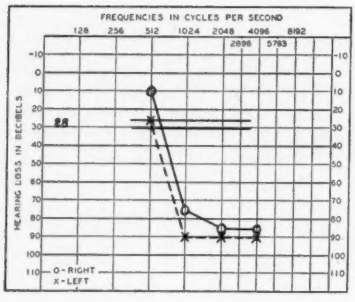

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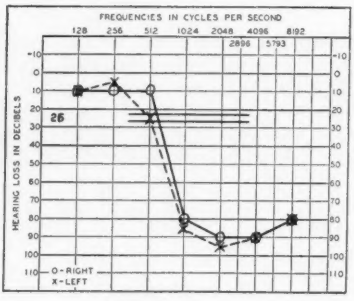

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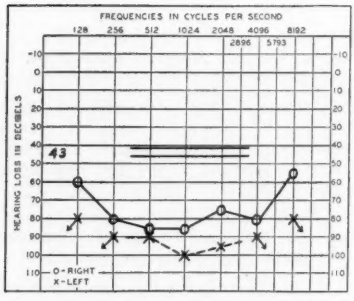
Fig. 2, Case 2.

CASE 2.—*CONTROL. Organic deafness, nerve type.* Age 24. After Army training as a machine-gunner, this man was sent overseas where he was in heavy combat on the Belgium-Holland border. He dates the onset of his tinnitus from the burst of a large-caliber shell which knocked him down. Shortly thereafter, he began to have trouble hearing ordinary conversation, although he could hear loud commands. He was hospitalized in France, treated for "shell blast" and sent to limited duty with an Air Forces ground-crew. Air conduction was better than bone conduction in both ears, with bone conduction markedly reduced; the sound of a 512 d.v. fork was lateralized to the left from the midline. The tympanic membranes were of normal appearance; there was a severe, constant, bilateral tinnitus, sometimes more severe in the left ear; some improvement in hearing was reported when the tinnitus lessened.

This patient had been demobilized six months prior to this examination. He seemed reasonably well adjusted as to economic and domestic circumstances, and reported the kind of hearing in group-conversational situations that is to be expected from the acoustic distortion represented in the pure-tone audiogram (Fig. 2*A*). He showed marked emotionalism during the recital of the onset of his hearing loss.

The Doerfler-Stewart test was negative. There was no evidence of impaired hearing prior to military service. The variation of 8 decibels in the speech-reception tests (Fig. 2*A* and *C*) is considered reasonable for this pattern of residual hearing. With the extreme drop-off of mid-tone and high-tone thresholds, and with the history of recent acoustic trauma, some variation in speech-hearing thresholds is to be expected.



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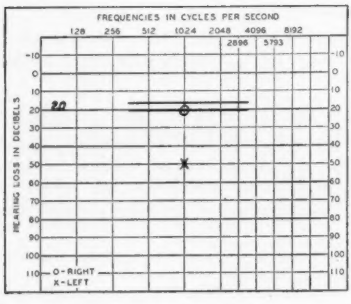

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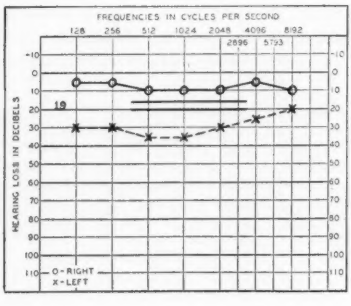

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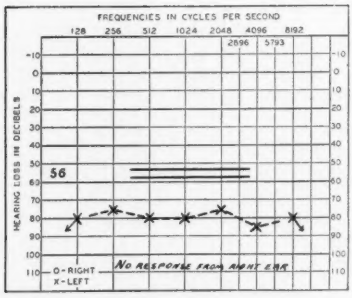
Fig. 3, Case 3.

CASE 3.—*Psychogenic overlay on a marginal unilateral impairment of undetermined origin.* Age 17. The history was negative except for an acute otitis media, three months previously, which had responded to treatment; the tympanic membranes were of normal appearance. Air conduction was greater than bone conduction in the right ear; bone conduction was greater in the left ear, and markedly reduced in both ears.

Routine tests and retests presented a bizarre picture. The 43-decibel threshold for speech reception (Fig. 3*A*) is disparate for the pure-tone thresholds. The Lombard test was inconclusive, but the Stenger was positive. The Doerfler-Stewart test was positive on three counts. On the day of the initial audiometric examination, hearing for a 1000-cycle tone at 60 decibels was admitted for the better ear, but denied when presented binaurally. Monaural-binaural speech-reception thresholds were equally disparate: the right-ear threshold was 20 decibels; the left ear 56 decibels; the binaural threshold was 44 decibels. There was no lateralization from the midline in tests with the 512 d.v. tuning fork.

The meaning of these findings was discussed fully with the patient, and opportunity was made for him to rationalize the recovery of useful hearing. He protested his innocence of malingering, although he freely admitted his discontent with military service. The possible relation between his environmental maladjustment and the recent ear infection was discussed, and he agreed to the use of suggestion hypnosis as a means to stabilize the test findings. A retest was made under excellent hypnotic control (Fig. 3*B*). The final test (Fig. 3*C*) was made after a period of eight days; the picture of normal hearing in the right ear and a marginal conduction type impairment in the left ear is consistent with the history and the findings.

Many clinicians would call this a clear case of malingering. There was ample evidence of marginal emotional conflict, however—quite enough to unsettle an immature person who finds himself in an unpleasant environment from which he cannot escape. In any event, it is believed that the clinical experience was salutary for the patient.



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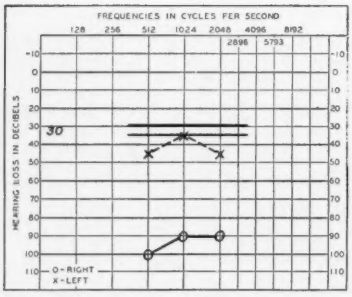

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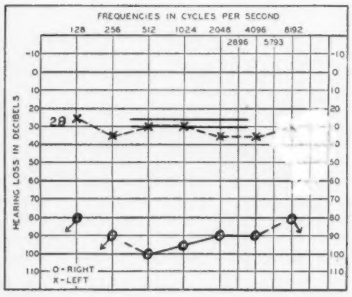

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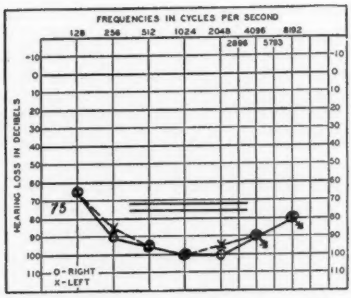
Fig. 4, Case 4.

CASE 4.—*Pyschogenic overlay on mixed type deafness.* Age 28. This veteran had been a sergeant in a demolition squad. The onset of impaired hearing was insidious, 40 months previously, with constant tinnitus in the left ear. There was a history of otorrhea in the right ear, following an acute otitis media 32 months previously. Examination of the tympanic membranes showed a large perforation of the posterior quadrant in the right ear, with a slight amount of purulent discharge; the left drum was slightly retracted. Tuning-fork tests produced the response of "vibrations only" in the right ear by bone conduction, with no "hearing"; in the left ear, air conduction was greater than bone conduction, with the bone conduction greatly reduced. There was no lateralization from the midline.

Attention is directed to the speech-reception threshold of 56 decibels (Fig. 4*A*). Monaural-binaural tests with headphones gave the following results: a 49-decibel threshold for the left ear; a 91-decibel threshold for the right ear; a binaural threshold of 51 decibels. The Lombard, Stenger and Doerfler-Stewart tests were positive.

This patient agreed readily to the use of suggestion hypnosis as a means of diagnosis. When the improved thresholds were determined (Fig. 4*B*), a strong suggestion of hearing recovery was employed. The findings were discussed in detail with the patient, and he seemed relieved to learn that he had a great deal of residual hearing and did not have to look forward to a profound physical disability. There were no readily apparent conflicts; he had a job in the family's business and expressed complete satisfaction with his prospects.

Repeat tests with the 512 d.v. tuning fork indicated matched air and bone conduction in the left ear, with bone conduction reduced; there remained a response of "vibration only" in the right ear. There is some doubt as to the finality of the pure-tone thresholds (Fig. 4*C*) which can be resolved only by follow-up tests as yet unavailable.



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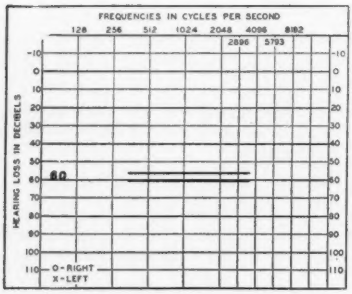

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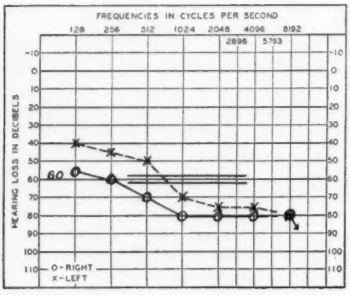


FINAL TEST

Fig. 5, Case 5.

CASE 5.—*Functional manifestations accompanying a severe mixed type deafness.* Age 19. This patient was a radio technician who gave a history of constant exposure to loud signals during which there frequently was an experience of tickle and pain in the ears. He stated that he first noticed an impairment for speech-hearing six months previously. Bone conduction was greater than air conduction in both ears, with the bone conduction reduced. The tympanic membranes appeared normal; there was constant bilateral tinnitus. There was no history of otic infection; his father was reported to be suffering from chronic otitis media and to being hard of hearing for several years.

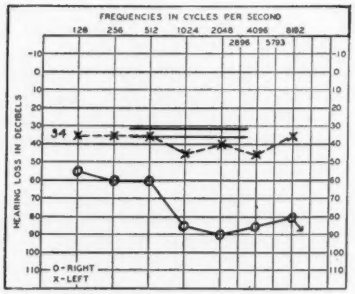
Seven retest audigrams showed a consistent improvement in pure-tone thresholds (the last of these being essentially the same as Fig. 5C) with the threshold of speech-reception intelligibility stabilizing at 60 decibels. The Lombard test was inconclusive. Neuropsychiatric examination indicated "a predisposition to functional involvement."

Two retests under suggestion hypnosis were made, with particular attention to the level of speech-reception intelligibility. The patient was fully cooperative, and control was excellent. In both these tests the speech-reception threshold was obtained at 60 decibels.

A following Doerfler-Stewart test indicated bilateral organic deafness with some evidence of a basic conductive type lesion.

Repeated consultations were employed to discuss the relations between organic involvement and the psychologic accompaniments of hearing disability. It was considered that the retest findings confirmed a severe, organic impairment, probably otosclerosis with nerve involvement. This patient was fitted with an air-conduction hearing aid which brought his speech-reception threshold to 24 decibels with relatively little distortion in sound discrimination.

The case illustrates the difficulty to be encountered in differentiating clearly between causal and accompanying psychologic factors in severe hearing disability.



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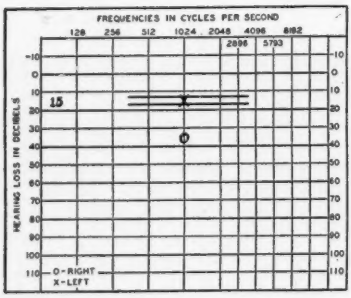

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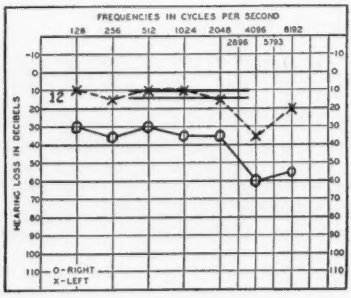


FINAL TEST

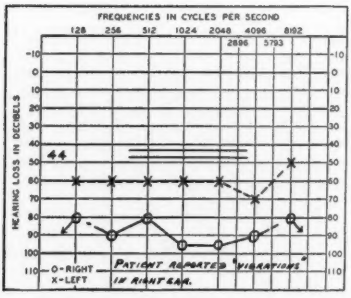
Fig. 6, Case 6.

CASE 6.—*Psychogenic overlay on mixed type deafness.* Age 19. This man was a private first-class, assigned to demolition. Although he had a history of chronic otitis media, he dated his hearing disability from an acute ear infection only eight months previously. On return to duty he found great difficulty in hearing commands and complained of a persistent headache in the frontal-temporal regions. He reported frequent exposure to blast, but no traumatic incident. There was intermittent bilateral tinnitus, more severe in the right ear. Air conduction was better than bone conduction in both ears, with bone conduction reduced; with the tuning fork at the midline there was lateralization to the left.

Retests elicited unstable responses, although the relationship between the two ears in pure-tone thresholds was fairly constant. The Doerfler-Stewart test was indeterminate. Monaural-binaural findings for speech reception were as follows: right—63 decibels; left—41 decibels; binaural threshold—36 decibels. These results are disparate and clearly represent confusion in the speech-hearing function.

A retest with the use of suggestion hypnosis was undertaken (Fig. 6*B*). Responses were prompt and definite. The final audiogram was done five days later (Fig. 6*C*).

This man was afraid of his work with demolition and afraid that he would lose his hearing. He had had trouble with his right ear since childhood and had suffered a slight high-tone hearing loss such as is typical of exposure to very loud sound. Psychotherapy was carried out to give him insight into his tendency to refer his anxiety to his ears.



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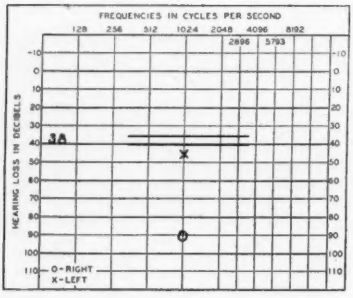

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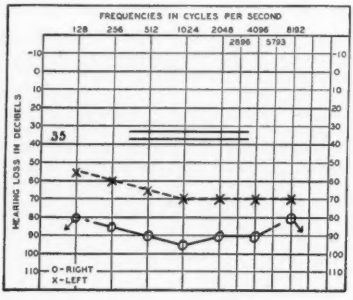


FINAL TEST

Fig. 7, Case 7.

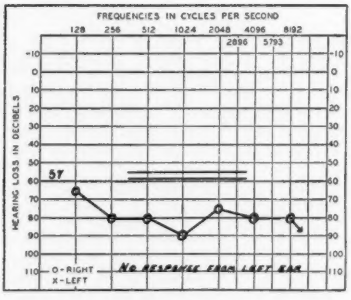
CASE 7.—*Psychogenic overlay on a nerve type involvement of undetermined extent.* Age 24. This patient had had six years of Naval service as an aviation mechanic, much of it aboard aircraft carriers in combat; he reported repeated exposure to heavy fire and blast. The onset of the hearing disability was insidious and dates back two years. The family history was negative; there was no history of tinnitus, and only an occasional headache. Air conduction was greater than bone conduction in both ears, with bone conduction much reduced; "vibrations only" were reported in the right ear, even by air conduction. His enlistment had expired and there was strong motivation to leave the service.

The correlation in the initial test between speech-reception and pure-tone thresholds was considered disparate (Fig. *7A*). The Lombard test was inconclusive; the Doerfler-Stewart test was positive on two counts.

Retest under suggestion hypnosis was carried out under conditions of good control. A 1000-cycle tone at 90 decibels in the right ear elicited a severe tonic spasm that awakened the patient. Several consultations were employed to help him understand the relations between his traumatic experiences (he reported having been knocked down five times by blast in the course of one engagement) and his hearing disability. He was fully cooperative and volunteered the information that a loud acoustic stimulus, particularly in the right ear, caused him to "remember" unpleasant combat experiences.

A second test under suggestion hypnosis produced the results recorded in Fig. *7B*. Follow-up audiograms were of the order of that shown in Fig. *7C*. The patient clearly understood that the pure-tone thresholds were considered disparate and that the speech-reception threshold was considered more nearly representative of the hearing loss. The patient was referred for psychiatric examination. There was no evidence of deep emotional conflict, and it was concluded that the most adequate treatment would be removal from the environment associated with traumatic situations.

The persistent, flat pure-tone thresholds shown in Fig. *7C* suggest a continued functional response. One can only conjecture that a "true" audiometric picture might show a classical pattern of nerve involvement from acoustic trauma, with the expected down-tilt of the high-tone thresholds, and with the right ear more severely affected than the left.



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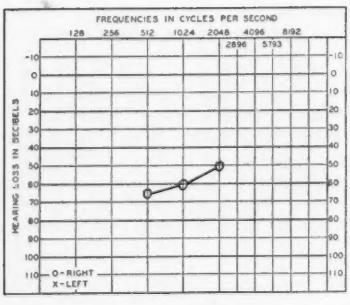

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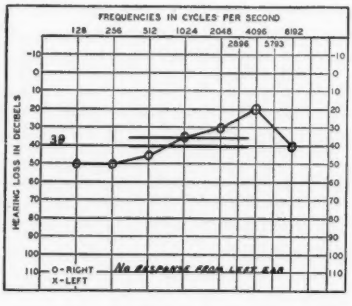


FINAL TEST

Fig. 8, Case 8.

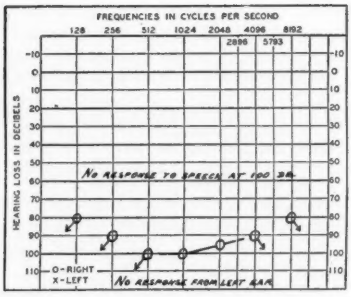
CASE 8.—*Functional overlay on organic disease, with the possibility of unilateral hysterical conversion.* Age 39. This veteran was receiving compensation for both hearing loss and psychoneurosis. He had had extensive combat duty, and stated that he first noticed a serious speech-hearing loss following a bomb-burst in France 30 months previously, which inflicted multiple shrapnel wounds and a severe concussion. He reported no dizziness, but constant tinnitus in the right ear. Bone conduction was better than air conduction in the right ear, and prolonged; there was no response to the 512 d.v. fork in the left ear, both with and without masking. Tests with cold caloric stimulation indicated an active vestibular mechanism on both sides. The family history was negative, and there was no history of otic infection prior to military service. The Wassermann test was 4-plus and the patient was undergoing antiluetic treatment.

Monaural-binaural speech-reception tests supported the findings shown in Fig. 8*A*: right—56 decibels; left—no response; binaural threshold—50 decibels. The Stenger test was positive, indicating hearing in the left ear. The Doerfler-Stewart test was positive.

This patient readily admitted spontaneous abreaction of combat experiences. He had tried two responsible jobs after his discharge from the Army but had been forced to resign "because the strain was too great." He insisted that he was happy with his wife and five children, but medicosocial field reports indicated that there had been one separation since his return home and that there were persistent disagreements with his wife over the reception and use of compensation checks. In overt manner, this patient was sauve and self-assured. He admitted to being disturbed by recurrent memories of his experiences in combat but admitted no other personal or family problems. He was relatively unperturbed about his hearing disability.

The use of suggestion hypnosis was unsuccessful on two occasions, but improved thresholds in the right ear were obtained under deep relaxation, as shown in Fig. 8*B*. At that time he denied hearing in the left ear, although the clinician noted signs of hearing.

The relations between his traumatic experiences and his hearing disability were discussed, and follow-up tests were stabilized at the level indicated in Fig. 8*C*. Tuning-fork tests supported the diagnosis of a conduction lesion in the right ear. The causal relations obtaining in the left ear were undetermined. It was considered that this patient had physical disabilities more serious than the hearing dysfunction, that there were deep neuroses present, and that further treatment was a matter for the psychiatrist to determine.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2896 5793
HEARING LOSS IN DECIBELS
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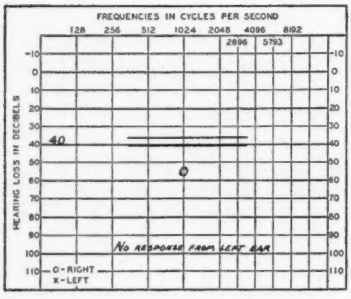
No response to speech at 100 db.
O-RIGHT
X-LEFT
No response from left ear

INITIAL TEST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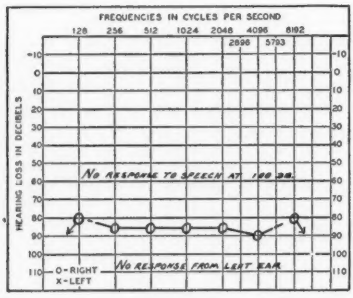
O-RIGHT
X-LEFT

TEST UNDER SUGGESTION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2896 5793
HEARING LOSS IN DECIBELS
-10 0 10 20 30 40 50 60 70 80 90 100 110
No response to speech at 100 db.
O-RIGHT
X-LEFT
No response from left ear

FINAL TEST

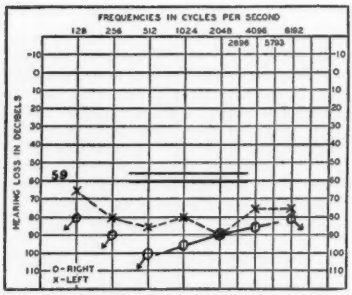
Fig. 9, Case 9.

CASE 9.—*Psychogenic overlay on organic deafness, with the possibility of a unilateral involvement.* Age 49. This man was a Navy veteran who had been fitted with a hearing aid three years previously. He had been exposed to heavy gunfire, and gave a history of acoustic trauma with a gradual return of auditory acuity; later, there was a fungus infection in both external ears, followed by acute bilateral otitis media. There was no history of tinnitus, but one of frequent, general headaches. Bone conduction was greater than air conduction in the right ear and greatly reduced; there was no response to the 512 d.v. tuning fork in the left ear, although caloric stimulation showed vestibular function.

As shown in Fig. 9*A*, there were no pure tones heard in the left ear. The patient denied any intelligible speech-hearing in the free-field test with the loudspeaker output at the maximum of 100 decibels. Yet, on the same day, a threshold for speech-hearing of 50 decibels was obtained with the use of headphones, the response coming from the right ear. The Doerfler-Stewart test was positive.

With the use of suggestion hypnosis the findings shown in Fig. 9*B* were made. These were supported by a consistent response, under hypnosis, to a very low conversational voice at 20 inches.

These findings were discussed with the patient and he seemed relieved to know that his "profound" hearing loss was not permanent. In audiometric retest, however, the response was similar to that in the first test (Fig. 9*C*); no free-field speech-reception threshold was obtainable, even at maximum amplification. This man had many problems of vocational and psychosocial adjustment and, because it was circumstantially impossible for him to remain under observation, he was referred to the neuropsychiatric department for outpatient treatment.



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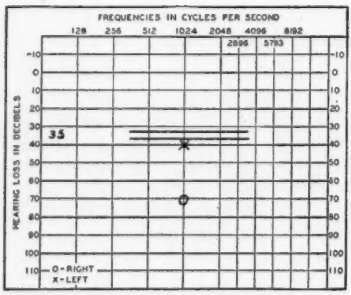

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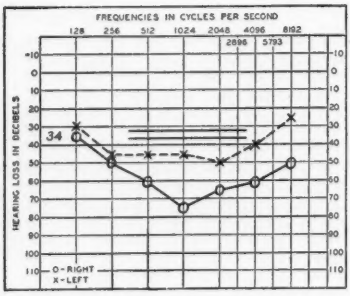


FINAL TEST

Fig. 10, Case 10.

CASE 10.—*Psychogenic overlay on organic deafness of undetermined origin.* Age 19. This patient reported exposure to practice gunfire about six months previously and dated his hearing loss from that time. Tympanic membranes were of normal appearance; tinnitus was reported to last for a day or so after exposure to very loud noise. The family history was negative; there was no history of otic infection. Air conduction was better than bone conduction, and the bone conduction reduced, in both ears. The sound of the 512 d.v. fork was lateralized to the left from the midline.

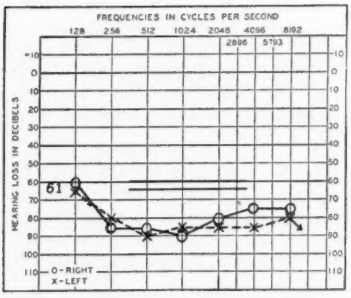
Initial test findings, shown in Fig. 10*A*, were considered disparate. Follow-up audiometry produced widely deviant pure-tone thresholds, and speech-reception thresholds varied between 35 and 40 decibels. The Stenger test was inconclusive. The Doerfler-Stewart test was positive. During the period of routine retest, the patient reported that he thought his hearing in the left ear was improving.

His general manner was restless and confused; he was vague and preoccupied in conversation. During the third consultation he admitted that six months previously a "friend" in his home-town had written that the patient's young wife had been flagrantly unfaithful and had become "the talk of the town." With a considerable emotional display, the patient insisted that he still loved his wife, said that they had been in correspondence, and that she had promised to be faithful to him in the future. It seemed obvious that some sort of aggravated physical symptoms would persist until this man's domestic affairs were in order.

Under suggestion hypnosis this patient responded as shown in Fig. 10*B*. While under hypnosis he responded readily to a very low conversational voice at a distance of three feet; he did not respond to the same voice at a greater distance.

The relation between the emotionally upsetting domestic affairs and the hearing disability were discussed fully, and the patient gave evidence of real insight into his reactions.

Follow-up tests with the tuning fork support the diagnosis of a nerve type involvement, with slightly more impairment in the right ear. Although the audiogram shown in Fig. 10*C* is not considered definitive, there is reason to suspect a congenital organic impairment.



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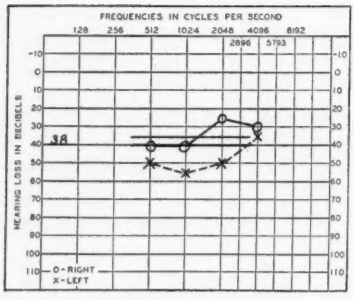

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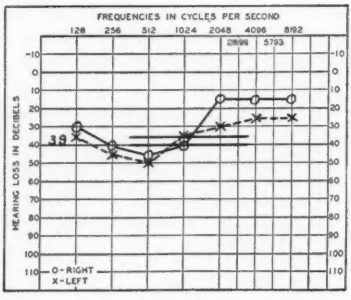


FINAL TEST

Fig. 11, Case 11.

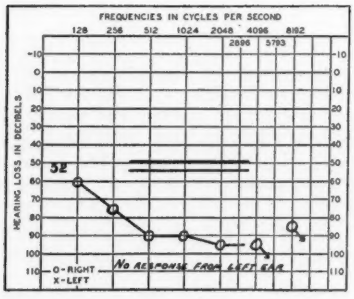
CASE 11.—*Functional overlay on conductive type deafness.* Age 23. This Army veteran dated his hearing disability back three years. He took part in four combat actions, but stated that his hearing had never completely gone. The family history was negative. The tympanic membranes were of normal appearance; there was a history of intermittent, bilateral tinnitus. He had attended college for the two years immediately previous, and recently found that his hearing loss was a genuine deterrent in all conversational situations. Air conduction and bone conduction were approximately equivalent in both ears, with the bone conduction normal; there was no lateralization with the 512 d.v. tuning fork at the midline.

The initial audiometric findings, shown in Fig. 11*A*, were considered disparate; retest four days later showed deviations. The Doerfler-Stewart test was positive.

During discussion of these test disparities, the patient reported that his wife was on the verge of a nervous breakdown and was under psychiatric care. She was hospitalized three days later.

The test findings shown in Fig. 11*B* were obtained with the use of suggestion hypnosis. These findings were discussed and the patient admitted that the combination of "expecting not to hear clearly" and nervousness about his wife's condition probably caused him to accentuate his hearing problems.

The test shown in Fig. 11*C* is considered representative of this person's organic hearing loss, which may be caused by otosclerosis.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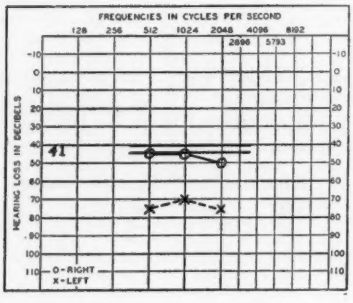
128 256 512 1024 2048 4096 8192
2896 5793
MEARING LOSS IN DECIBELS
-10 0 10 20 30 40 50 60 70 80 90 100 110
32
O-RIGHT
X-LEFT
No response from left ear

INITIAL TEST



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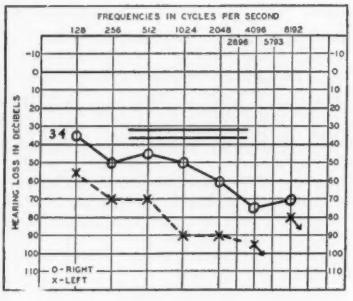

FINAL TEST

Fig. 12, Case 12.

CASE 12.—*Psychogenic overlay on nerve type deafness.* Age 25. This Army veteran wore a hearing aid in his right ear, from which he was gaining very little benefit in speech hearing. He dated his hearing loss back two years, in connection with machine-gun fire in combat. Both tympanic membranes showed unhealed perforations, dating from that time; there was constant bilateral tinnitus. Air conduction was greater than bone conduction in the right ear, with the bone conduction reduced; there was a minimal bone conduction response in the left ear; the 512 d.v. tuning fork was referred to the right from the midline.

The findings of the initial test were considered disparate (Fig. 12*A*). The Doerfler-Stewart test was positive. Retest under suggestion hypnosis produced the results shown in Fig. 12*B*. The disparities were discussed with the patient and he freely admitted a feeling of emotional tension and insecurity. There was serious illness in his family, and he was quite uncertain of his ability to hold his job. The general impression he gave was one of a rather immature, hysteroid personality.

On returning a month later, this patient produced test responses quite similar to those of his first test. He was retested once again under suggestion hypnosis with precisely the same results that had been obtained previously. A follow-up test, done the same day as this second retest with hypnosis, produced the results shown in Fig. 12*C*. This audiometry is believed to represent the level of the organic impairment, and the patient was scheduled to be refitted with a more appropriate hearing aid.

DISCUSSION

Some effort has been made to achieve a differential diagnosis by means of technique involving galvanic skin responses.[6] This technique with the use of a standard electroencephalograph seems to have merit in making a gross distinction between organic and psychogenic deafness, but, as recently reported, the responses are not sufficiently refined to indicate the extent of the psychogenic involvement. Unless this extent can be determined, the clinician knows little more than he would learn from a careful study of test-retest data.

Hurst[4] and Semenov[10] discuss unsuccessful attempts to use suggestion hypnosis in working with psychogenic deafness; because no mention was made of amplification, it is assumed that amplification was not employed in making contact with the patient.

The question is sometimes raised as to the suggestibility of the patient, for he must be suggestible in order that suggestion hypnosis may be used. Both reason and experience indicate that most persons with a psychogenic involvement in hearing disability are more or less suggestible. A deep level is not necessary for auditory testing.

As one becomes aware of the possibility of psychogenic deafness as a clinical entity, sustained by the presence of bizarre inconsistency in routine auditory tests, he realizes that he is faced with an unknown element in the patient's responses. The usefulness of suggestion hypnosis as a diagnostic technique is that it can reduce, even quantify, that unknown element, and therefore help by that much to clarify diagnostic thinking. This is a worthwhile contribution to a confused and confusing clinical picture. Whether the practising otologist uses deep suggestion or whether it is incorporated into the diagnostic and treatment procedures of a large medical center, is beside the point. The essential point is that the otologist must be alert for the "unknown element" that controls the deviant clinical data. He is the specialist who must rule on the presence or absence of organic involvement and assume responsibility for a clear diagnosis. As time goes on, with the accumulation of data from the otologist in the field, the definition of psychogenic deafness should become a simpler matter.

SUMMARY

Some of the problems in the diagnosis and treatment of psychogenic deafness are discussed and emphasis is given to the fact that the most common finding is a psychogenic overlay on organic disease. Because this is true, a special diagnostic requirement is the ability to measure accurately the relative amounts of organic and psychogenic

involvement. The burden rests directly upon the otologist, for he is the specialist most capable of interpreting hearing tests and of evaluating the otic pathology. The use of several special tests in the clinical work-up is discussed, and suggestion hypnosis is recommended as a simple means of measuring the organic and functional components.

Twelve cases, two of which are controls, are presented to illustrate the use of these techniques. Of special importance is the ease with which pure-tone and speech audiometry can be used with suggestion hypnosis for diagnostic purposes. The findings with deep suggestion are authenticated by follow-up tests. The key to the therapy, once the proportion of organic involvement is known, is the ability of the patient to gain insight into the relations between the causal factors and the symptoms. Under many circumstances this phase of the treatment can often be more readily accomplished by the otologist than by the psychiatrist.

JOHNS HOPKINS HOSPITAL.

REFERENCES

1. Brenman, M., and Gill, M. M.: Hypnotherapy, New York, Macy Foundation Review Series, 1944.

2. Doerfler, L., and Stewart, K.: Malingering and Psychogenic Deafness, J. Speech Disorders 2:181-186 (Sept.) 1946.

3. Harbert, F.: Functional and Simulated Deafness, U. S. N. Med. Bull. 41: 458-471, 717-728 (March, May) 1943.

4. Hurst, A. F., and Miller, E.: Discussion on Functional Deafness, J. Laryng. & Otol. 55:393-404 (Aug.) 1940.

5. Martin, N. A.: Psychogenic Deafness, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 55:81-89 (March) 1946.

6. Michels, M. W., and Randt, C. T.: Galvanic Skin Responses in the Differential Diagnosis of Deafness, Arch. Otolaryng. 45:302-311 (March) 1947.

7. Nash, C. S.: Psychosomatic Manifestations—Otolaryngology, Trans. Am. Acad. Ophth. & Otolaryng., November-December, 1947.

8. Rennie, T. A. C.: Psychosomatic Manifestations—General Aspects, Trans. Am. Acad. Ophth. & Otolaryng., November-December, 1947.

9. Rosenberger, A. I., and Moore, J. H.: Treatment of Hysterical Deafness at Hoff General Hospital, Am. Jour. Psych. 102:666-669 (March) 1946.

10. Semenov, H.: Psychic Deafness, Trans. Am. Acad. Ophth. & Otolaryng., March-April, 1947.

11. Thompson, E. A., Hardy, W. G., and Pauls, M. D.: Hearing Disabilities and Aural Retraining, in: Looseleaf Medicine of the Ear, E. P. Fowler, Jr., ed., New York, Thomas Nelson & Sons, 1947.

12. Truex, E. H., Jr.: Psychogenic Deafness, Connecticut State M. J. 10:907-915 (Nov.) 1946.

VI

INFLUENZAL LARYNGITIS

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Acute inflammatory edema involving primarily the larynx, when caused by Haemophilus influenzae, type B, invariably has a rapid onset and may require emergency tracheotomy. Symptoms vary primarily with the extent and degree of submucosal edema and cellular infiltration. This may range from moderate swelling to complete filling of the hypopharynx within a period of a few hours. Direct or indirect laryngoscopy and early accurate bacteriological identification are the two essential procedures in the early recognition of this disease. Streptomycin is the newest addition to our armamentarium in combating this disease process. Six cases are reported.

In 1936 Le Mierre[1] reviewed the French and German literature regarding septicemia due to Pfeiffer's bacillus. All cases reported up to that time were divided into two types; primary septicemia following local infection of the larynx or the pharynx with occasional complicating endocarditis; and septicemia occurring during a "grippy" course in severe cases of influenza, with Pfeiffer's bacillus recovered at postmortem examination from bronchopneumonic areas in the lungs. All cases of the first type were in infants or children and one out of six recovered. No specific therapy had been used up to that time.

In this same paper Le Mierre also gave the first reported case of this infection with laryngeal symptoms and septicemia due to this organism in an adult. His patient was a 49-year-old female who had chills, fever and acute illness of 24 hours' duration. Dyspnea, laryngeal obstruction and pharyngitis with edema of the anterior surface of the epiglottis, and abscess of the cervical area with positive culture

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of Pfeiffer's bacillus from both the blood stream and the cervical abscess were noted. Complications of arthritis of the left shoulder, pharyngeal ulceration, anemia and loss of weight occurred during the period of positive blood stream findings. Recovery required one month. No therapy is described.

The English literature provides several additional papers which deal primarily with the same clinical picture as seen in infants and children. In 1941 Sinclair[2] reported four deaths in a series of 10 cases in infants and children and obtained a postmortem examination of one case. There was involvement of the epiglottis, cords, tracheal and primary bronchial mucosa. He used sulfonamide therapy in addition to tracheotomy and supportive measures. One patient who developed a complicating meningitis recovered with the aid of type-specific anti-serum. Jordan and Turner[3] used type-specific anti-serum in three cases and lost a fourth patient treated with sulfadiazine and penicillin. A second case in an adult was added by McLorinan[4] in 1946 in a report of 16 cases with two deaths.

Between March and August 1947 six cases of edematous laryngitis due to H. influenzae, type B, were encountered at the Massachusetts Eye and Ear Infirmary. Of the six cases four occurred in adults, bringing the total of adult cases to six thus far reported.

The pathological findings, very similar to those originally reported by Le Mierre, were well illustrated in a 19-month-old infant who, on initial examination, was not breathing and was pulseless. Onset of symptoms occurred ten hours prior to death. The throat and blood cultures were positive for Haemophilus influenzae, type B. No treatment had been received prior to examination. Death was attributed to acute respiratory obstruction.

Postmortem findings included marked swelling of the epiglottis and aryepiglottic folds with [illegible] swelling of the ventricular bands, and still less swelling of the true cords and tracheal mucosa as seen on gross examination (Fig. 1). Sections of the epiglottis stained with hematoxylin-eosin showed marked edema and cellular infiltration which was submucosal in location. Study of serial sections of the larynx showed that the inflammatory reaction decreased from the epiglottis downward with minimal inflammatory reaction subglottically. Sections stained with Glynn's tissue stain for bacteria showed numerous gram-negative bacilli consistent with the morphological characteristics of H. influenzae, the majority being located deep submucosally. The primary pathology involved the epiglottis both clinically and at postmortem examination, and substantiated

Fig. 1, Case 1.—Larynx and trachea showing subglottic extension of inflammatory reaction without exudate.

the misnomer of acute epiglottitis often used in describing this disease.

These findings present an interesting contrast to the laryngeal, tracheal and bronchial necrosis observed in cases of acute laryngotracheobronchitis due to streptococcal infection by Schall.[5]

The five remaining patients in the present series recovered and are living and well, having had no recurrence of symptoms. From five to 63 years of age they all complained of sore throat, dysphagia and respiratory distress of short duration. They all had low-grade fever, yet looked much sicker than the fever or other clinical findings would indicate. There was moderate leukocytosis, positive throat cultures for H. influenzae, type B, and negative smears and cultures for Klebs-Loeffler bacilli. All showed marked inflammatory edema of the larynx and especially of the epiglottis on direct and indirect laryngoscopy (Fig. 2). Hoarseness was not a constant symptom. Blood cultures were obtained in two cases and were positive for H. influenzae, type B. Lateral x-ray films of the neck were obtained in all cases and showed marked swelling of the epiglottis in each case.

Bacteriological studies were carried out utilizing aerobic and anaerobic broth and blood agar cultures. The organism failed to grow in the broth and required blood for propagation. Positive identification of the organism was obtained by the Quellung reaction using type-specific anti-serum as described by Alexander.[6] He uses a loopful of type-specific H. influenzae, type B, anti-serum on a slide. To this he adds one loop of methylene blue and one loop of cultured organisms. He reports that the capsular swelling of the organism is positive within 30 minutes. A rapid typing method utilizing the above technique but substituting nasopharyngeal secretions was subsequently described by the same author.[7] He emphasizes that morphologically H. influenzae and pneumococci in mucus are often indistinguishable in methylene blue preparations without the aid of type-specific anti-serum.

Abscess of the epiglottis which did not require incision and which resolved during treatment, delayed complete resolution of the epiglottic swelling, and hoarseness with slowly resolving ventricular band swelling were the only complications encountered. No meningitis, arthritis, cervical abscesses, endocarditis, toxic myocarditis, empyema or manifest tracheobronchitis as reported by other investigators was observed.

Many patients seen initially are hospitalized with a presumptive diagnosis of diphtheria, and during the past year several cases of clinical diphtheria without membrane have been seen. The illness is of longer duration and immediate smears and study of five-hour cultures on Loeffler's medium are done on hospital admission to rule out this possibility.

Acute edema of the larynx caused by other gram-negative organisms may closely simulate this picture especially when Bacillus coli is the predominating organism. Three such cases were encountered during the same period as those reported. All responded well to sulfadiazine or streptomycin and none required tracheotomy. Angioneurotic edema due to food allergy and foreign body, such as a fishbone in the vallecula which impinges on the epiglottis each time the patient swallows, can produce markedly similar symptoms.

Retropharyngeal abscess in infants presents a longer history and the physical findings are characteristic. Parapharyngeal abscess of otitic origin may present swelling of the epiglottis and hypopharynx as a secondary manifestation, but the positive otologic history and the marked unilateral cervical, nasopharyngeal and pharyngeal swelling serve to aid in differentiation.

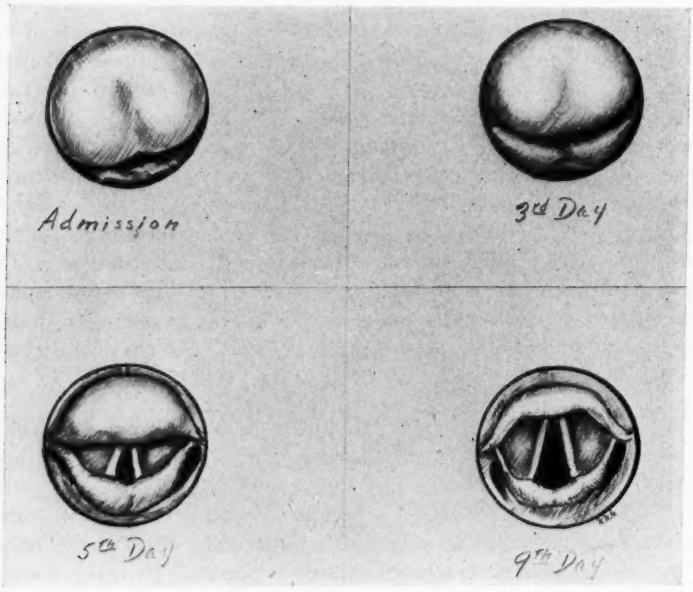

Fig. 2, Case 5.—Serial indirect laryngoscopic findings.

Comments.—The six cases presented are similar with regard to history, clinical findings and laboratory findings to those previously described in the literature as representing infections due to Haemophilus influenzae, type B, with septicemia following primary laryngeal and pharyngeal involvement.

The untreated case from which microscopic sections were obtained appears to be the first case in which histopathological findings are illustrated.

The first patient to be treated with streptomycin was given sulfadiazine and penicillin initially, and symptoms and epiglottic swelling began to recede, remained stationary for two weeks and then recurred. Rapid resolution was felt to be the result of intensive streptomycin therapy, the patient receiving three grams per day for seven days.

Of the treated cases one child and one adult had an entirely uncomplicated course. Two patients developed abscess of the epi-

glottis. Though symptoms cleared rapidly following treatment and the swelling was quickly reduced at first, complete resolution of the epiglottic inflammatory edema required nearly a month after discharge from the hospital.

The oldest patient, aged 63 years, had a normal appearing epiglottis on the thirteenth day, but apparent abscess of the ventricular bands necessitated continuation of the use of the tracheotomy tube until the thirtieth day. Following hospital discharge subsequent slow resolution, in view of the patient's age, suggested readmission for biopsy of the false cords. The pathological report was chronic inflammation with no evidence of carcinoma or gram-negative bacilli found in the sections studied.

No evidence of toxicity or sensitivity attributable solely to streptomycin was observed.

The following mode of investigation and therapy in patients giving a history of sore throat, dysphagia and increasing respiratory distress of short duration is suggested.

1. Immediate tracheotomy if necessary, aided if possible by bronchoscopic or straight Mosher life-saver airway for relief of marked dyspnea with retraction and as a guide to the trachea.

2. Mirror laryngoscopy in adults and direct laryngoscopy, if necessary, in infants. Often the swollen epiglottis can be seen on firm depression of the base of the tongue.

3. Lateral film of the neck to rule out the presence of a foreign body.

4. Smears and cultures for Klebs-Loeffler bacilli.

5. Rapid typing of nasopharyngeal secretions by Alexander's technique.

6. Blood culture.

7. Streptomycin, 0.5 to 1.0 gram per day in children depending upon age and 2.0 grams per day in adults, given parenterally in divided doses.

8. Penicillin given parenterally to combat secondary coccal invaders.

9. Fluids given parenterally to maintain fluid and salt intake in the presence of dysphagia and dehydration.

10. Humidified room to prevent respiratory obstruction from crust formation, especially following tracheotomy.

SUMMARY

1. Histopathological study of a case of Haemophilus influenzae, type B, infection of the larynx and pharynx with septicemia is described.

2. Four additional cases in adults are reported, bringing the total to six.

3. Five cases of acute edema of the larynx treated with streptomycin are recorded.

4. Sudden onset of sore throat, fever, dysphagia and hoarseness with mild to severe respiratory distress suggest the possibility of this type of clinical picture.

5. Positive diagnosis is based primarily on laryngoscopy, lateral cervical radiography and accurate early bacteriological diagnosis.

6. Streptomycin appears to be of definite value and more specific toward Haemophilus influenzae, type B, than any previously available therapeutic agent.

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REFERENCES

1. Le Mierre, A., Meyer, Andre, and Laplane, R.: Les septicemies à bacille de Pfeiffer, Ann. de Med. 39:97 (Feb.) 1936.

2. Sinclair, S. E.: Haemophilus Influenzae Type B in Acute Laryngitis with Bacteremia, J. A. M. A. 117:170 (July 19) 1941.

3. Jordan, R. B., Jr., and Turner, J. R.: The Therapy with Antiserum of Acute Laryngitis Caused by Haemophilus Influenzae, J. Ped. 27:572-575 (Dec.) 1945.

4. McLorinan, H.: A Report on Sixteen Cases of Supra-glottic Oedema, M. J. Australia 1:220-222 (Feb.) 1946.

5. Schall, L. A.: Personal communication.

6. Alexander, H. E.: Type "B" Anti-Influenzal Rabbit Serum For Therapeutic Purposes, Proc. Soc. Exp. Biol. & Med. 40:313 (Feb.) 1939.

7. Alexander, H. E.: Craig, H. R., and Ellis, C.: Validity of Etiologic Diagnosis of Pneumonia in Children by Rapid Typing from Nasopharyngeal Mucus, J. Ped. 18:31 (Jan.) 1941.

VII

THE FISSULA ANTE FENESTRAM OF THE HUMAN OTIC CAPSULE

II. ABERRANT FORM AND CONTENTS

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In the preceding article the authors discussed the developmental anatomy of the fissular and surrounding portions of the otic capsule in man, and the adult form of the fissula with its normal contents. In the entire investigation, 300 series of sections of fetal, postnatal and adult temporal bones were examined.

By way of introduction, it is important to stress the fact that ossification of the otic capsule begins during the sixteenth week of intra-uterine life. Thereafter centers of ossification appear in rapid succession and fuse to produce an almost complete osseous capsule in the 22-week fetus.[5] The fissular region, however, is slow to ossify and appears as a considerable mass of cartilage with fibrous core in the fetus of 21 weeks (183 mm.), in which it extends from the cartilaginous rim of the vestibular window to the cochlea, forming a considerable part of the posterolateral wall of the latter structure. The contained seam of connective tissue, which occupies the fissular seam, passes through the lateral wall of the capsule from the tympanic to the vestibular surface. External to the cartilage is an osseous shell, which by accretion becomes part of the general petrous mass. The cartilage is progressively reduced in bulk by slow conversion into intrachondral bone. The association of fibrous, chondral and

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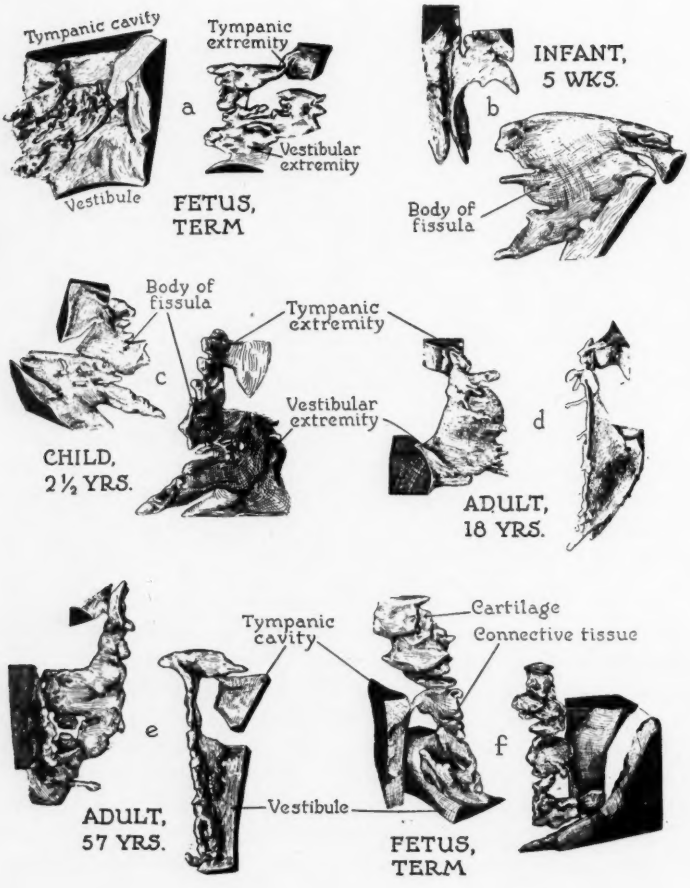

Fig. 1.—Drawings of wax-plate reconstructions of the space of the fissular channels; selected series, depicting variation in form, size and contents; two views of each (adapted from Anson and Martin[3]). In most of the figures only small portions of the tympanic cavity and the vestibule have been included; in the lefthand figure of 1*a* and in both figures of 1*f* larger portions of each are included, together with the intermediate area of the vestibular window. X 12.

osseous tissues is thus established at the midfetal stage: connective tissue is embedded in cartilage and the latter is lodged in bone. Thereafter, and normally throughout life, the structural pattern is retained, being modified only in respect to proportionate bulk of the constituent elements: cartilage is reduced in amount and bone is increased, connective tissue remaining the while but slightly altered in amount.

NORMAL FORM AND CONTENTS

The fissular portion of the otic capsule in the adult ear is made up of an assemblage of tissues. Within the periosteal layer primary bone persists as circumferential lamellae, and a similar endosteal layer commonly forms a delicate pellicle. Calcified cartilage is present in the form of islands scattered throughout the endochondral trabeculae; the periphery of a cartilage cell lacuna is gradually rendered osseous, with subsequent obliteration of the space by bone tissue and incorporation into the adjacent endochondral structure. Connective tissue proper occurs as several fibrous stripes within the walls of the otic capsule, and one of these is the fissula ante fenestram. Lastly, unaltered hyaline cartilage is found; it is normally restricted in the adult to the vestibular orifice of the fissula and to the adjacent vestibular window.

The fissula ante fenestram makes its appearance at the 34-mm. (9-week) stage as a connective tissue invagination on the anterior wall of the vestibule at the point of junction with the scala vestibuli. It extends anteriorly toward the cochlea, then rostrally to turn at a right angle lateralward when it reaches the horizontal level of the vestibular (oval) window. By the 75-mm. (12-week) stage it has reached the tympanic surface of the capsule in or below the semicanal for the tensor tympani muscle.

In postnatal life the form of the fissula may be that of an irregular wing, as in the 5-week infant (Fig. 1*b*); however, the outline is more frequently that of a crescent or an open letter "S," as in the 18-year adult (Fig. 1*d*). Prolongation of either the anterior or the posterior margin may occur either toward the cochlea (2½-year child, Fig. 1*c*) or in the opposite direction toward the vestibular window (term fetus, Fig. 1*a*). Whereas, in the typical fissula, the body narrows gradually in its ascent (Fig. 1*d*), the superior aspect may be abruptly constricted (57-year-old adult, Fig. 1*e*).

In all of the above instances the uppermost part of the fissula extends to, or but slightly beyond, the transverse level of the tym-

panic orifice; and the body of the fissula, whether pronged (Figs. 1*a* to 1*c*) or alar (Fig. 1*d*), is compressed from side to side. However, in a large number of cases the fissula extends cranialward beyond the level of the tympanic orifice (Fig. 1*f*) or, while not greatly exceeding average height, it is capacious enough to assume a columnar form and thus to cause the upper portion of the fissula to appear as a cupola-like extension. The cupola may be associated with a typically narrowed caudal portion (Fig. 1*f*) or with one of equal caliber. It is in fissulae belonging to one of these latter categories, or in those possessing combined features, that cartilage masses are likely to occur.

The extremities of the fissula are likewise subject to variation. Infrequently, the tympanic orifice is lacking, or it may open into the semicanal for the tensor tympani muscle while the vestibular orifice reaches the scala vestibuli. Yet, despite variations, the tympanic orifice is invariably situated in a more craniad position than the vestibular and it is almost without exception the smaller of the two orifices. Occasionally, the vestibular orifice is of relatively reduced size. It is sometimes crossed by bridges of bone, indicating that once established in the embryonic otic capsule both its shape and size are subject to alteration by subsequent growth of the surrounding osseous tissue.

Variability in form of the fissular channel is dependent upon the arrangement of adjacent histologic constituents. Whereas, in typical instances, the fissula is merely a narrow cleft in the otic capsule with tympanic and vestibular orifices, it frequently communicates also with the vestibular window. It may open without interruption across the window from the regular site of the vestibular orifice to that of the tympanic, thereby bringing into continuity periotic, ligamentous, and submucosal connective tissues. In specimens with an unusually capacious fissula, the immediate wall of the tract is a perichondrium covering adjacent cartilage, at the periphery of which is situated intrachondral and endochondral bone with related marrow spaces. This is the histologic succession encountered regularly in the fissula, whether large or small. Even when the otic capsule is undergoing dramatic and rapid conversion from cartilage into bone, the perichondrium remains intact and potentially active. In typical cases, during late fetal life, the perichondrium is associated with a thin lamina of cartilage continuous with similar tissue on the vestibular and tympanic walls. In cases of a large fissula, part or all may become occupied by cartilage.

Subsequently, such newly formed cartilage undergoes conversion peripherally into endochondral bone. Rarely is there replacement by vascular bone of the sclerotic type. The presence of cartilage and bone within a fissular tract would seem to represent stages in the reconstruction of the otic capsule which had not attained histologic maturity. Its instability, then, would appear to depend upon its structural incompleteness.

ABERRANT FORM AND CONTENTS

In spite of its small size, great interest attaches to the histologic fate of the fissula ante fenestram because it is situated in the territory most frequently occupied by otosclerotic bone. Small as is the fissula, the vestibular (oval) window is but little larger. As a consequence, any deforming effect which the exuberant production of otosclerotic bone may have on the fissular area may ultimately be exerted on the neighboring window. It is generally believed that when newly formed bone, by encroachment on the fenestral space, reduces the excursion of the stapes, loss of hearing acuity occurs.

While the commonest site of occurrence of otosclerotic bone is in the antefenestral area, it is well known that these pathologic foci are encountered elsewhere in the labyrinthine capsule and that continuity may not exist between their separate sites of origin.* However, the scope of the current report will be restricted to the normal and abnormal transitions in histologic structure of the fissula ante fenestram and to a comparison between this entity and certain other sites of delayed ossification in the temporal bone.

Since residual cartilage or defective bone is frequently found in fetuses and young children in the region of the fissula, it is of interest to first examine sites of residual cartilage in other areas of the capsule.

Residual Cartilage. Residual cartilage occurs in seven different regions in fetal and infantile temporal bones: the fossula post fenestram; the postero-inferior portion of the cochlear capsule, or infracochlear region; the basal portion of the styloid process; the petrosquamous suture, and the capsule beneath the suture; the canalicular region; the region of the cochlear (round) window; and the fissula ante fenestram.

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*Independent foci of otosclerotic bone have been observed to involve the cochlear (round) window by Habermann,[19, 20] Schilling,[39] Manasse,[28] Panse,[35] Siebenmann,[41] Bruhl,[11] Mayer,[31, 32] Wittmaack,[44] Lange,[26] Brunner,[12] Grunberg,[17] and Hagens;[21] the facial canal by Hartmann,[22] Bruhl,[9] Mayer,[30] and Ruttin;[37] the region of the internal auditory meatus and base of the cochlea by Politzer,[36] Haber-

Cartilage masses, either wholly chondral or partially ossified, are infrequently found in the region of the fossula post fenestram, having been observed in only five per cent of those ears bearing fossulae. Since they are rare and since the fossula itself is not a constant structure, this area possibly is of little importance in relation to pathology of the petrous bone. However, Ruttin[37] described a focus of otosclerotic bone situated near "a large absorption space directly behind the niche of the oval window, filled with connective tissue"; it is interesting to note that this pathologic focus adjacent to the fossula post fenestram involved the posterior margin of the stapes as well.

A mass of residual cartilage is found in the infracochlear portion of the capsule in about 26 per cent of late fetal petrous bones. In a larger percentage of late fetal bones there occurs in this region a bar of endochondral bone surrounded by the periosteal bone of the otic capsule. Since this cartilage mass usually disappears at term or in early infancy, it is not likely to be related to subsequent pathological processes.

The styloid and adjacent bony process develop as a rule from two distinct cartilages, which remain unchanged for some time after the otic capsule becomes well ossified. By the third postnatal year, however, they have generally undergone complete ossification.

Similarly, in the region of the petrosquamous suture residual cartilage masses are found in 33 per cent of full-term fetuses and in 29 per cent of infantile petrous bones. In a few cases a center of poorly formed, gnarly bone occurs in the place of the cartilage rem-

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mann,[20] Bruhl,[10] Manasse,[28] Mayer,[30] Siebenmann,[41] Wittmaack,[44] Eckert,[15] and Kosokabe;[25] the cochlear capsule by Siebenmann,[40] Manasse,[27] Alexander,[1] Bruhl,[11] Wolff,[46] Lange,[26] Kosokabe,[25] Doderlein[14] and Wittmaack;[45] the modiolus and laminae by Alexander;[1] and the region of the semicircular canals by Siebenmann,[40] Moller,[33] Bruhl,[11] Mayer,[31] Nager,[34] and Hagens.[21] An otosclerotic focus in the apex of the pyramid was described by Wittmaack.[44] More recently Guild[18] described 113 otosclerotic foci in 81 pathological temporal bones; these areas were distributed anterior to the oval window in 65 (of 81 ears), at the anterior margin of the round window in 25, in the stapedial footplate (primary area only) in 10, in the cochlear capsule in 13 (anterior part, 6; inferior part, 5; antero-inferior part, 1; superior part, 1), in the fundus of the internal auditory canal in 2, and in the cochleariform process in 1 case. Rarely have otosclerotic foci been described outside the labyrinthine capsule. Katz[23] observed otosclerotic patches in the malleus and incus of a cat, and Covell[13] described a similar pathologic process in human ossicles. In 1943, Keleman[24] described a unilateral otosclerosis-like focus forming the wall between two pneumatic cells in the immediate vicinity of the cortex of the tegmen antri. Concerning this case, Shambaugh[38] stated that, "There is a question whether such areas of new bone occurring outside the labyrinthine capsule should be called otosclerosis, since they probably have nothing to do with the well-defined clinical and pathologic entity known as otosclerosis."

nants. Since, in most cases, ossification is complete in this region before birth and since the percentage of residual cartilage is almost as great in infants up to two years of age as in term fetuses, the condition suggests an abnormal delay in ossification. In older infants the occasional centers of poorly formed, gnarly bone indicate a late attempt at conversion of this residual tissue into a more mature cellular structure.

Although the otic capsule is largely ossified at birth, isolated foci of cartilage persist in the canalicular portion of the capsule in about 65 per cent of the petrous bones from late fetuses and infants up to three years of age. Actively growing cartilage is found in 10 per cent of cases; what its fate may be is at present unknown. In the remaining 90 per cent a quiescent type of cartilage is observed. Otosclerotic foci in the canalicular region usually occur in the endochondral capsule immediately surrounding the semicircular canals. However, in the authors' specimens, residual cartilage in the canalicular region lies outside the endochondral bone, either in the arch of the superior canal, or, more frequently, lateral to that site. In one of Siebenmann's[40] cases otosclerotic foci are situated directly at the endosteum and in the center of the planes of the semicircular canals.

Growth of the cartilaginous otic capsule is negligible after midfetal life, but a certain degree of expansion continues in the cartilaginous portion along the basal and posterior margins of the cochlear (round) window after the remainder of the capsule has ossified. This growing cartilage which aids in delimiting the cochlear window ossifies late. However, it does so in a normal manner and no residual cartilage has been observed at this site in late stages. Otosclerotic foci are, nevertheless, commonly found at the periphery of the cochlear window.

In a considerable number of late fetal and infantile ears histologic changes occur in the area of the fissula ante fenestram which are of an aberrant nature. These changes consist of a chondrification of part or all of the fissular connective tissue, either as a circumscribed mass or as a neoplastic nodule which may spread beyond the bounds of the fissula into the adjacent capsule. This secondary cartilage requires more detailed description.

Secondary Cartilage. The masses of secondary, or proliferating, cartilage apparently arise from the dormant cartilaginous pellicle situated at the margin of the fissula (see Fig. 6 of the preceding article[4]). In some instances the new cartilage is limited to but one portion of the fissula, replacing some of the original connective tissue (Fig. 2);

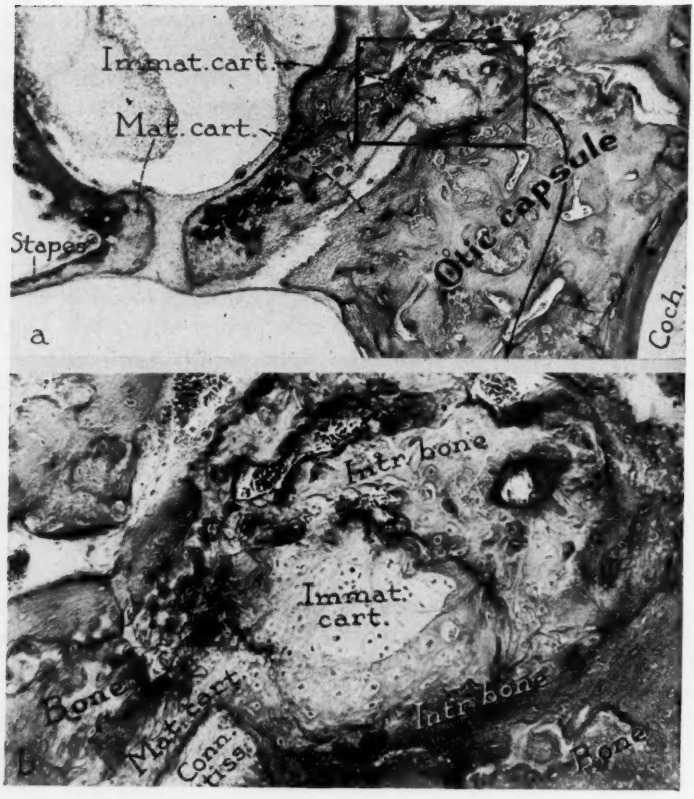

Fig. 2.—Photomicrographs (horizontal sections) of the vestibular orifice of the fissula ante fenestram and a cartilage nodule (chondroma) situated near the cochlear end of the fissula; infant of 6 months (Northw. ser. 10.11.35). Fig. 2*a* includes the related cochlea, tympanic cavity, and vestibule. X 39. Fig. 2*b* records the difference between the normal cartilaginous shell and the pathological tissue of the chondroma in the area indicated in Fig. 2*a*. X 180.

Abbreviations for these and the succeeding figures: *Ann. lig.*, annular ligament (of stapes); *Ant. crus*, anterior crus (of stapes); *Bl. v.*, blood vessel; *Calcified cart.*, calcified cartilage; *Cart.*, cartilage; *C.* or *Coch.*, cochlea; *Conn.*, *Conn. tiss.* or *Conn. tissue*, connective tissue; *Fenest. orif.*, fenestral orifice of fissula ante fenestram; *Immat. cart.*, immature cartilage; *Intrach. bone* or *Intr. bone*, intrachondral bone; *Mat. cart.*, mature cartilage; *S. or Stap.*, stapes; *T.* or *Tymp. cav.*, tympanic cavity; *T.o.*, or *Tymp. orif.*, tympanic orifice (of fissula); *V.* or *Vest.*, vestibule; *V. o.*, *Vest. orif.* or *V.o.f.a.f*, vestibular orifice of fissula ante fenestram.

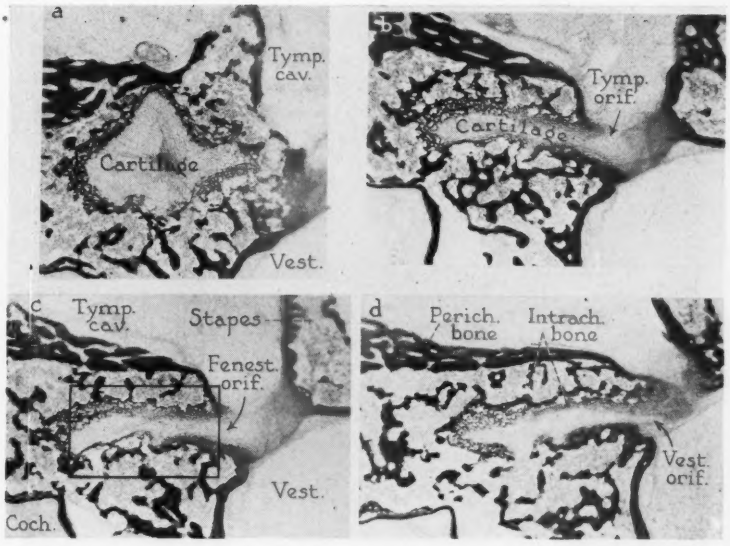

Fig. 3.—Photomicrographs at four levels through a chondromatous mass occupying the fissula ante fenestram, in a 180-mm. (21-week) fetus (Wisc., ser. 45B). Fig. 3*a*, cranial to the tympanic orifice; *b*, at the latter opening; *c*, at the level of the fenestral orifice; *d*, at the vestibular extremity. X 25.

in others it involves the entire fissular tract (Figs. 3 and 4). Such replacement occurs commonly in the late fetus, infant and child, at ages when ossification of the otic capsule should have been completed. Of 27 series of temporal bones from term fetuses, infants, and children up to 10 years of age, the fissular tract is occupied by a cartilaginous mass in 16 cases. The cartilaginous masses of secondary formation appear for the first time in older fetuses[7]; the earliest stage of occurrence is in a 180-mm. (21-week) fetus. The manner of origin and spread and the mechanism by which such cartilage is subsequently reduced in bulk or replaced by a more active tissue will now be considered.

The new cartilage that comes to occupy a fissular channel containing fibrous tissue is derived from the perichondrium which regularly lines the channel.[7] In all such cases there is a gradual histologic transition from perichondrial connective tissue to young cartilage. In a 180-mm. fetus, the occurrence of such a precociously developed

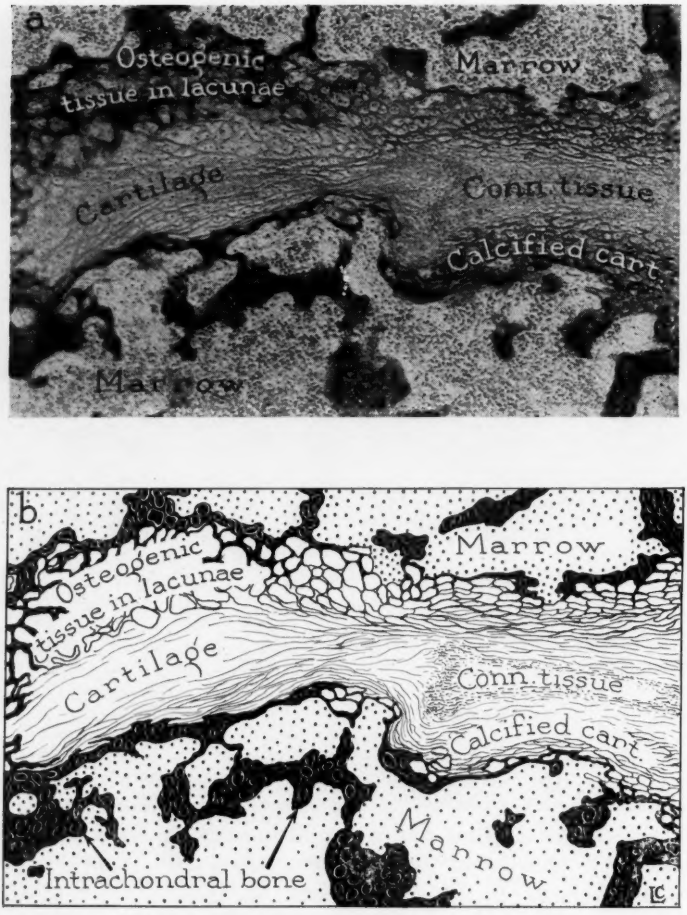

Fig. 4.—Photomicrograph and drawing showing the details of histological structure of a cartilage mass in the fissula ante fenestram, from the area outlined in Fig. 3*c*; *a*, photomicrograph; *b*, same field diagramed, showing marginal encroachment by intrachondral bone and obliteration of connective tissue by neoplastic cartilage. X 95.

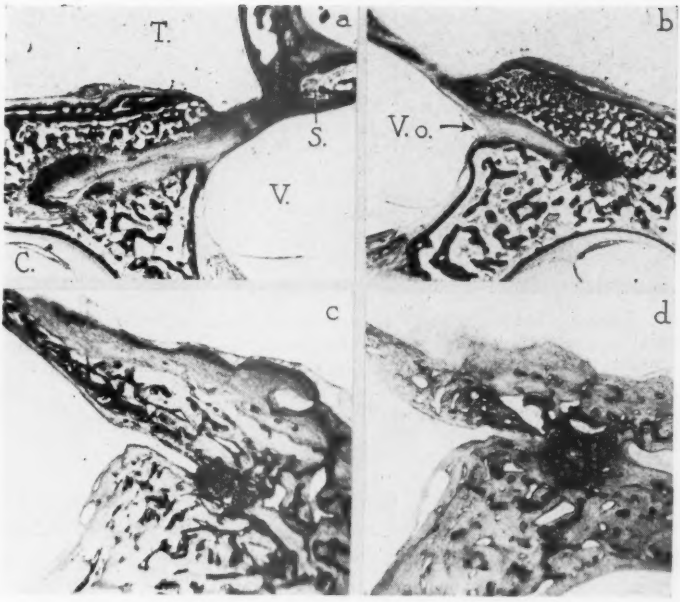

Fig. 5.—Photomicrographs of the fissular region of the otic capsule (Bast[7]), showing cartilage situated near the cochlear margin; *a*, 180-mm., 21-week fetus (Wis. ser. 29B); *b*, 202-mm., 23-week fetus (Wis. ser. 70); *c*, 3-year infant (Wis. ser. 96B); *d*, 10-week infant (Wis. ser. 83). X 16.

cartilage within the fissular space makes clear the manner in which the new tissue gradually displaces the original connective tissue of the fissula to produce a chondromatous mass within the bone between the cochlea and the vestibular window. Craniad to the tympanic orifice the new cartilage fills a cupola-like extension of the fissular space (Fig. 3*a*) and reaches the tympanic orifice (Fig. 3*b*). At the fenestral communication it leaves a mere seam of connective tissue (Fig. 3*c*) and pushes toward the orifice (Fig. 3*d*). Marginally there is a gradual transition from perichondrial connective tissue into newly formed cartilage (Figs. 4*a* and 4*b*). Therefore, the cartilage in many young specimens occurs not only as a parietal investment for the connective tissue of the fissula but also as a nodular mass which partially or wholly replaces that tissue.

In the cartilage nodule the cells are not arranged in orderly fashion as they are in mature "quiescent" hyaline cartilage. The

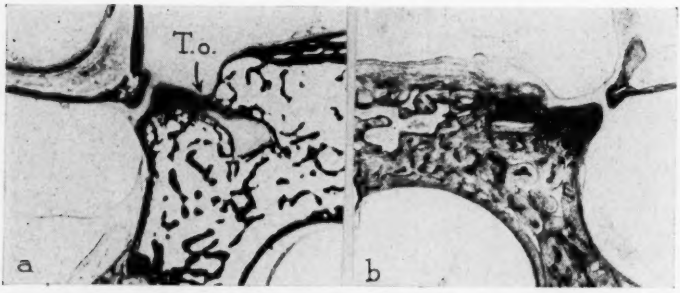

Fig. 6.—Photomicrographs of the fissular region (Bast[7]), illustrating ossification occurring between the tympanic portion of the fissula and the stapedial articulation; *a*, 222-mm., 25-week fetus (Wis. ser. 46); *b*, 14-month infant (Wis. ser. 93). X 13.

tissue of the nodule, having replaced the fibrous portion of the fissula, experiences alteration within its own mass (Figs. 4*a* and 4*b*). Once formed, its matrix undergoes tinctorial change, staining deeply with hematoxylin in the manner of cartilage prior to invasion by osteogenetic buds. Apparently this cartilage has reached the final stage preceding the process of ossification. Supportive evidence for this opinion is offered by the older ears, in which the cartilage mass is being slowly replaced by osteoid or even by intrachondral bone.[7] Thereafter, the cartilage is not invasive. It does not produce atrophy of, to subsequently replace, the bony tissue of the capsule but merely succeeds a more primitive mesenchymal tissue.

The secondary cartilage which appears as a chondromatous mass in the antefenestral region of the otic capsule may be large or small, limited to any portion of the fissula or spread throughout its full extent. These masses of actively growing cartilage will now be considered on a regional basis.

Changes Along the Cochlear Margin of the Fissula.—These changes apparently occur in unusually deep fissulae (Fig. 5*a*, from a 180-mm fetus in which the fissula extends from the vestibule almost to the cochlea). At the anterior extremity of the fissula the surrounding cartilage has partly changed to the large-cell type and is deeply stained by hematoxylin. In a 202-mm. (23-week) fetus, the formation of the cartilage mass is well advanced (Fig. 5*b*), while in a three-year-old child a similar mass is undergoing ossification around the margin (Fig. 5*c*). In a 10-week infant the mass is ossified (Fig.

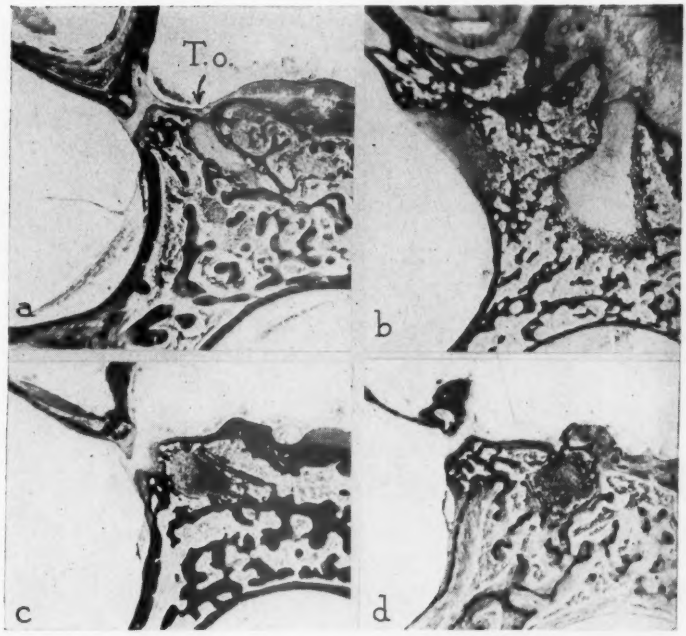

Fig. 7.—Photomicrographs of the fissula region of the otic capsule (Bast[7]) illustrating changes occurring at the tympanic extremity of the fissula without involvement of the stapedial joint; *a*, 265-mm., 29-week fetus (Wis. ser. 69); *b*, 260-mm., 29-week fetus (Wis. ser. 45B); *c*, 290-mm., 32-week fetus (Wis. ser. 59); *d*, 280-mm., 31-week fetus (Wis. ser. 116A). X 13.

5*d*). In such instances, the fissula is not obliterated by the process but merely compressed.

Changes Between the Tympanic Orifice of the Fissula and the Stapedial Joint.—In the region between the anterior margin of the vestibular window and the tympanic orifice of the fissula, the cartilage normally ossifies late, with a marginal layer of cartilage persisting for a variable length of time. Occasionally, in late fetal life, this area undergoes ossification, usually with the formation of intrachondral bone (Fig. 6*a*). Later, these islands may be surrounded by endochondral bone, as in a 14-month infant (Fig. 6*b*), or they may be re-excavated and partially refilled by endochondral bone.

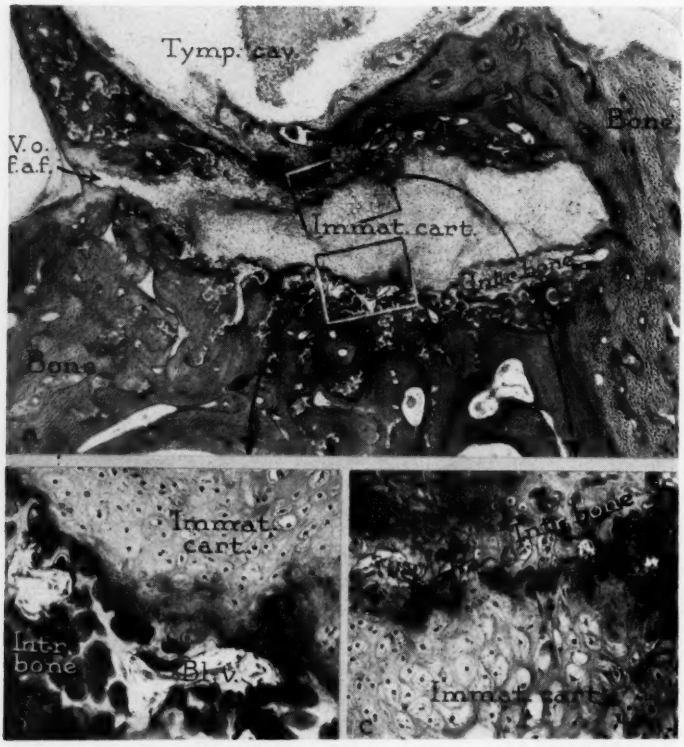

Fig. 8.—Photomicrographs of the vestibular orifice of the fissula and a chondroma occupying the latter, in an infant of 6½ months (Northw. ser. 8.11.38). *a* shows the relation of the cartilage mass to the tympanic cavity, cochlea, and vestibule. X 37. *b* and *c* record the detail of the areas outlined in *a*; they include the immature cartilage of the chondroma, the marginal intrachondral bone and the vascular channels. X 163.

This process sometimes extends beyond bounds to involve the fissula, either narrowing or obliterating it.

Changes at the Tympanic Orifice.—In many specimens there is partial or complete replacement of the fibrous content of the fissula at or near its tympanic orifice by actively growing cartilage or, in older specimens, by newly formed bone. This process of occlusion may result either from downward growth of the cartilage mass from the cupola-like extension of the fissula or from the independent formation of a nodule of active cartilage at the site of the tympanic orifice (Figs. *7a* to *7d*). In these instances, the large tympanic orifice is not reduced in the normal process of ossification, but subsequently by the expedient of new cartilage formation. Frequently the mass obturates the entire tympanic orifice. The chondromatous mass is sometimes removed by an appreciable distance from the vestibular window (Fig. *7b*), or it may lie very near the latter (Fig. *7c*). It more often occupies a position intermediate between these extremes (Figs. *7a* and *7d*).

Changes at the Vestibular Orifice.—When the vestibular end of the fissula is blocked by a mass of cartilage arising in situ, it is susceptible to the same histologic alterations affecting secondary cartilage in other sites (Figs. 8*a* and 9*a*). As pointed out in the initial discussion of secondary cartilage, these masses are often present early, being a striking feature even in some midterm specimens (Figs. 3 and 4). In the fetal capsule, when marrow is abundant but endochondral spicules are still sparsely distributed through the region of the fissula, the regular succession of tissues is already established within and immediately around the fissular channel. Young cartilage predominates, containing only a narrow seam of persistent fibrous tissue (Figs. 4*a* and 4*b*); the cartilage passes imperceptibly at its periphery into a calcified form of the same tissue, and the latter into intrachondral and endochondral bone. Such a fissula differs from the normal only in the amount of cartilage present, since, when the fissula first acquires an osseous wall, the cartilaginous stratum is normally represented by a thin perichondrium (Fig. 6 of preceding article[4]; 210-mm. fetus). In the infant (Fig. 8) and in the child (Fig. 9), the succession of tissues is the same; histologic change affects almost exclusively the surrounding capsular elements. In these older stages ossification has advanced to the point at which the fissula is supported by numerous trabeculae of intrachondral and endochondral bone (Fig. 9*a*) or by a more solid skeletal framework Fig. 8*a*). The newly formed cartilage approximates the intrachondral bone (Figs. 8*b*, 8*c*, and 9*b*) or the calcifying cartilage (Fig. 9*b*).

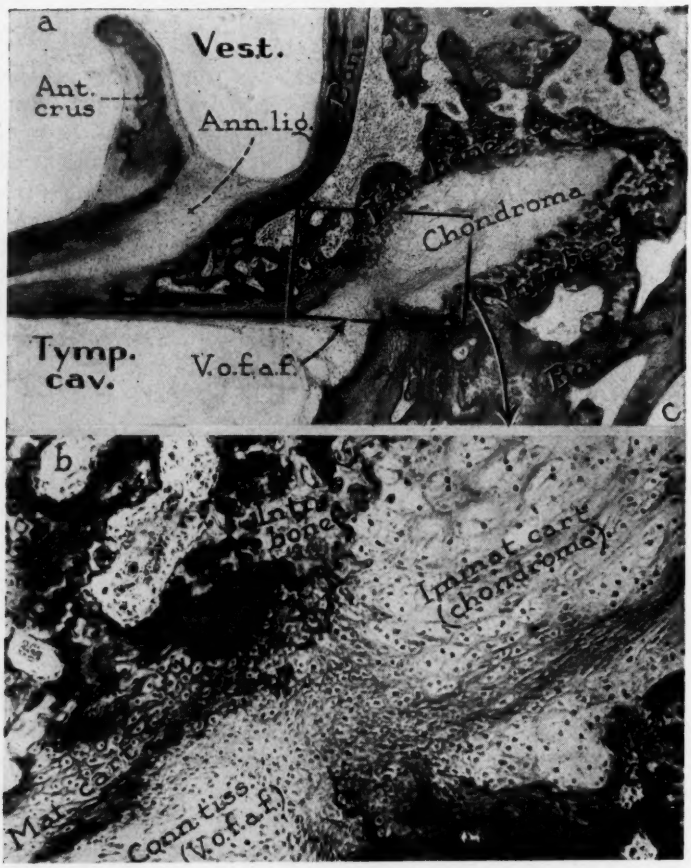

Fig. 9.—Photomicrographs of the fissula, with a contained chondroma, in child of 2 years (Northw. ser. 10.26.32). *a* shows the cartilage nodule extending toward the vestibular orifice of the fissula. X 39. *b* records histological detail in the area outlined in *a*. The active cartilage of the chondroma rests upon intrachondral bone; at the vestibular orifice connective tissue blends with the mature (quiescent) cartilage. X 165.

The histologic structure of the cartilage nodule and of its neighboring tissues remains fundamentally alike, whether the former lies deep within the vestibular portion of the fissula (Figs. 2*a* and 2*b*) or occupies the greater part of the entire vestibular extremity (Figs. 8*a* to 8*c*, 9*a* and 9*b*).

Comparably, the cellular character of the nodule persists essentially unaltered in older specimens. In fact, this tissue in late childhood and during adulthood is histologically indistinguishable from the newly formed cartilage of an infantile or fetal specimen. However, the peripheral tissues undergo modification; the intrachondral bone is slowly reduced in relative bulk through the progressive increase in quality of endochondral bone in the antefenestral area.

Changes Above the Tympanic Orifice.—Chondromatous nodules of the sort just described are quite often encountered in cupola-like extension of the fissula (Fig. 1*f*; see also Anson and Wilson,[2] Fig. 13, and Bast,[7] Figs. 48 to 50). The cartilage formed therein is apparently formed in an effort to reduce the space of an abnormally large fissular channel; the cartilage replacing the connective tissue is sequestered in the aberrant fissula as a result of delayed capsular ossification.

Changes Involving the Entire Fissula.—In a few specimens the cartilage occupies the entire extent of an enlarged fissula, extending without interruption from the supratympanic cupola to the vestibular orifice (see Anson and Wilson,[2] Fig. 14; Anson and Martin,[3] Figs. 52 to 56). Likewise, in a small number of cases, cartilage masses fill remnant spaces in fissulae whose orifices have become separated from the main, or middle, portions of the fissula by focal ossification. The new cartilage is histologically identical whether it be present as a unit or as multiple nodules.

The cartilage mass characteristically presses beyond the free surface of the tympanic wall (Fig. 7*b*) and at the vestibular extremity pushes toward the orifice, in which, however, some of the original connective tissue is likely to remain (Figs. 2*a* and 9*a*). At each extremity of the mass there is a gradual transition from perichondrial connective tissue into new cartilage. Between the latter and the surrounding intrachondral bone, a sharp line of demarcation indicates the marked difference in age of formation. The intrachondral bone, on the other hand, passes gradually into the older cartilage of the fissular wall, of whose perichondrial stratum it is a derivative. Complete transitional stages are found between the three tissues, each becoming continuous by imperceptible gradations with

the contiguous layer. A similar series of transitional stages is found at the numerous points at which new cartilage encounters marrow spaces and bulges into them (Fig. 8*b*). As described in the preceding article, the cartilage which lines the fissular tract and against which the new cartilage mass abuts is of a different order. It is a mature tissue which has been present in this locality since the stage of embryonic formation. Likewise "normal" are the islands of intrachondral bone which intervene between the mass of young cartilage and endochondral bone wherever the cartilage is not adjacent to marrow tissue.

Quite regularly the active cartilage which thus arises from the investing fissular cartilage undergoes partial ossification at a later stage. This replacing tissue may be either of intrachondral or endochondral character, the type of bone possibly depending upon the age of the subject at the time of formation of the mass. Thus, if the mass of active cartilage develops early, at a time when ossification of the cartilaginous capsule is usually completed, the formation of intrachondral bone occurs in an irregular fashion at the periphery of the mass (Fig. 7*b*). If the cartilage develops later, osteoid tissue may be formed (Fig. 7*d*). Still later, such masses may be excavated by vascular osteogenetic buds and replaced by endochondral bone. In instances in which the processes are delayed, as in early childhood, the islands of intrachondral bone become surrounded by a mixture of tissues comprised of osteogenetic buds, osteoid and patchy accretions of endochondral bone.

These various changes indicate that the fissular area is histologically deficient and unstable. The changes which take place in the cartilage nodules in early infancy frequently resemble those which occur in the production of otosclerotic bone. Later attempts at rebuilding this superimposed bony tissue, by excavation and redeposition of vascular, gnarly bone, serve to enlarge the original area. However, at the present stage of the study, the authors do not yet have available a sufficiently large number of specimens from children and adolescents to complete accurate observations on these sequential histologic alterations.

Sclerotic Bone. When sclerotic bone occurs in the antefenestral area, it may partially or wholly obliterate the fissular channel. Where replacement is partial, the vascular osseous tissue merges with cartilage of the chondromatous type. Thus, in a specimen from an 18-year-old adult, the fissular area is remarkable for the manner in which it terminates conventionally below but widens above into a relatively

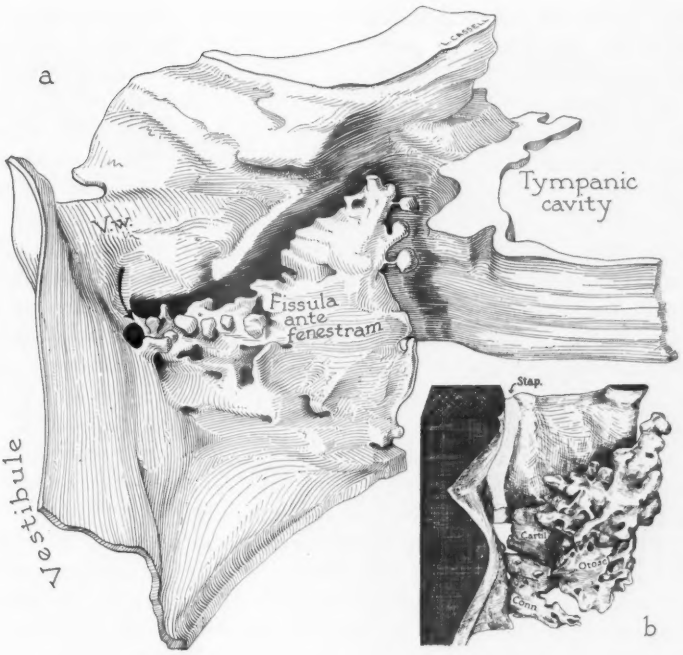

Fig. 10.—Drawings wax-plate reconstructions of the fissular tissues.

Fig. 10*a* includes the fibrous tissue of the fissular channel and similar tissues continuous therewith, namely, the periotic layer of the vestibule, the ligament of the stapes, and the submucosa of the tympanic cavity. Child of 10 years (Northw. ser. 11.28.30). Reconstruction by James W. Pick, M.D. Medial view. X 22. The vestibular extremity of the fissula ante fenestram is elongate and is prolonged antero-inferiorly to the roof of the scala vestibuli; the fenestral extremity (at arrow) is very very small; the tympanic extremity is out of view (hidden by the body of the fissula).

Fig. 10*b*, connective tissue, cartilage and sclerotic bone of the fissula, extending in that succession from the vestibular orifice upward into a cupola-like space situated cranially to the level of the tympanic extremity (Fig. 61 of Anson and Martin[3]). Adult of 18 years (Northw. ser. 1.17.31). Side view. X 17. Arrows point to the tympanic and vestibular orifices. of the fissula.

large nodule of sclerotic bone; a cartilaginous seam intervenes between the two dissimilar portions (Fig. 10*b*). Its communication with the vestibule is elongate and irregularly broken by areas of continuity between adjacent walls (fenestrations in the reconstruction). The fibrous tissue gives way to cartilage and the two tissues fuse through a narrow band of perichondrium. The preponderant part of the mass is composed of a vascular, spongy bone of the otosclerotic type, which extends cranialward and anteriorly toward the cochlear wall beyond its tympanic and vestibular connections. The mass is markedly irregular, bearing numerous projections which invade adjacent marrow spaces. At the tympanic extremity the sclerotic bone is exposed to the mucous membrane without the intermediation of cartilage or connective tissue. Here the relatively minute orifice lies at the bottom of a cone-shaped indenture and is bordered by mature cartilage which forms part of the investment of the vestibular window, and inferiorly extends along the vestibular wall and into the vestibular orifice of the fissula. In the specimen illustrated the general outline of the fissular content (Fig. 10*b*) is strikingly similar to that of a normal fissula (Fig. 10*a*); however, the rostral and intermediate parts of the fissula are more capacious, where sclerotic and chondromatous tissues, respectively, replace the fibrous content of the channel. The new cartilage and vascular bone of the pathological specimen (Fig. 10*b*) apparently serve, in the course of capsular development, to resolve an expansive fissular channel: cartilage replaces connective tissue and is in turn transformed into bone.

When sclerotic bone is present in limited amount, the fissular tract retains its familiar configuration (Fig. 10*b*). The sclerotic bone, in association with a cartilage nodule, then occupies but a part of the fissular space (in Fig. 10*b* the superior two thirds). However, when the new bone spreads beyond the fissular area, it may form a part of the capsular wall in the region of the vestibular window, replacing the cartilage originally present there (Figs. 11*a* to 11*e*). The new bone in the specimen illustrated is virtually limited to the antefenestral area, in the lateral wall of the capsule. It consists of a major anterior portion from which lesser root-like extensions reach partially around the superior and inferior margins of the vestibular window (Fig. 11*c*). The whole sclerotic area measures approximately 3 mm. in height (Fig. 11*d*). When the area is reconstructed as a separated enucleated mass, that surface which is directed toward the middle ear is found to be quite smooth and largely exposed to form part of the tympanic wall anterior to the vestibular window

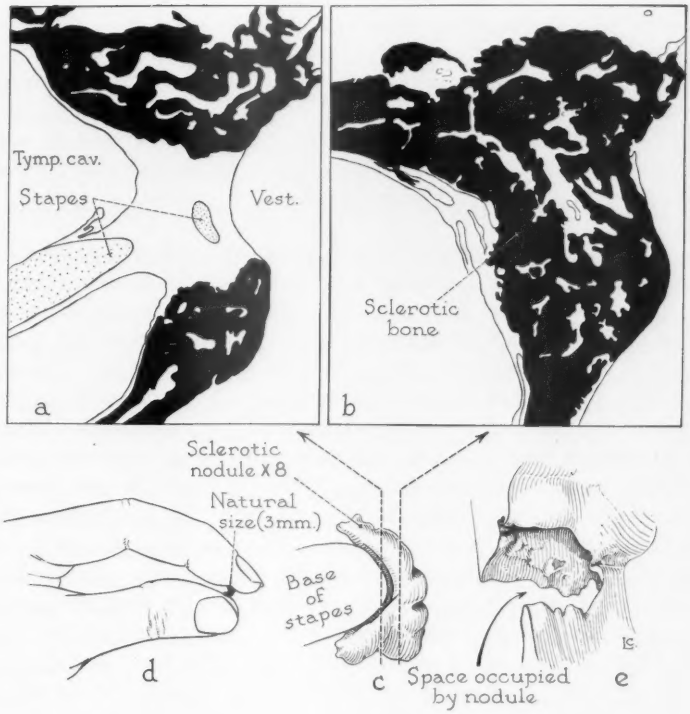

Fig. 11.—Drawings (vertical sections) through the spongious bone of an otosclerotic nodule and drawings of wax-plate reconstructions of the nodule and the surrounding area of normal bone; from an adult (Northw. ser. 12.6.29). Fig. 11*a*, section through the portion which borders upon the superior and inferior margins of the vestibular window at the latter's anterior end (contiguous normal bone omitted). Fig. 11*b*, section through a more anterior portion of the otosclerotic mass, just in front of the vestibular window (see levels indicated in *c*). X 26. Fig. 11*c*, reconstruction of the nodule of otosclerotic bone shown as if enucleated from the normal bone (*e*) in which it was lodged, and as seen from the medial, or vestibular, aspect. Fig. 11*d*, reconstruction of a portion of the capsule in which the nodule was situated, and from which the mass has been removed to leave an "excavation." X 8. Fig. 11*e*, the nodule, shown at natural size in relation to the size of the hand. X ½.

The photomicrographs demonstrate that the newly formed bone is vascular and porous and that it has completely replaced the normal osseous and cartilaginous tissues in the fenestral region; the figures of the reconstructions, enlarged, emphasize the latter feature and also demonstrate the way in which the pathological tissue is imbedded in the normal; the figure of the nodule held by hand records the diminutive proportions of the area of sclerotic bone.

(Figs. 11*c* and 11*e*); but much of the deeper aspect, directed toward the labyrinth, is irregularly lobulate and less exposed, with the lobulations producing corresponding excavations in the normal bone (Fig. 11*e*). Prolongations of the sclerotic mass form the boundary of the vestibular window in its anterior half but the stapedial ligament is uninvolved. The basal part of the mass is exposed to both tympanic and vestibular surfaces, except at its anterior limit where it is exposed only on the tympanic side.

In sections, the newly formed bone stains deeply acidophilic and can be readily distinguished macroscopically from the surrounding uninvolved bone. The rarefied bone consists of moderately well-defined lamellar systems arranged around prominent vascular spaces Figs. 11*a* and 11*b*; see also Anson and Wilson,[2] Pls. I and IV; Bast,[6] Fig. 36). The line of demarcation with normal bone is quite distinct and crenated in outline. The total absence of intrachondral bone in the spongy nodule and its abundance in the adjacent endochondral bone serves to further distinguish the old from the new tissue.* The central cavities within the sclerotic bone are larger than the haversian canals in the normal bone. These cavities contain a fine network of reticular tissue, which usually support only a single thin-walled vessel; anastomoses are effected with similar vessels wherever the containing cavities intercommunicate. In the zone of the root-like prolongations, blood vessels which enter and leave the sclerotic bone are related principally to the haversian vessels. Those which supply the main mass, bordering upon the vestibule and the tympanic cavity, are principally of periosteal origin.

This mass has encroached upon and obliterated all but the vestibular portion of the fissula ante fenestram. Not only has the fibrous content of the original fissular tract been replaced but the normal cartilage and bone of its periphery as well. This outcome is to be contrasted with the result of the spread of a chondromatous mass. The latter is confined to the fissular channel and to such marrow spaces as communicate therewith. Fissular connective tissue is replaced by the secondary cartilage, but the mature lining cartilage and the peripheral osseous shell undergo little or no reduction in amount.

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*The obliteration of calcified cartilage islands (intrachondral bone or cartilaginous interglobular spaces) at the site of otosclerotic bone has been commented upon by Siebenmann,[40] Schilling,[39] Wolff,[46] Manasse,[29] Fischer,[16] Anson and Martin,[3] Wilson,[42] and Bast.[8] Wittmaack[43] mentions the persistence of interglobular spaces in trabeculae of endochondral bone involved in the spongified bony area.

CONCLUSIONS

The antefenestral region of the human otic capsule is regularly crossed by a cleft of perilymphatic nature, the normal content of which is a slightly vascular connective tissue. This cleft, the fissula ante fenestram, is typically narrow and usually no greater in vertical height than the distance between its two regular orifices: one upon the tympanic cavity anterior to the vestibular window, the other into the vestibule at the site of junction with the scala vestibuli.

Frequently in fetuses, infants, and children, the fissula is unusually large. In such cases, it contains a mass or nodule of newly formed cartilage derived from the chondral wall of the fissula. Such a cartilage mass may occupy part or all of the fissular tract and may reach the vestibular window when an auxiliary, or fenestral, orifice occurs. The new cartilage mass forms at the expense of the original connective tissue. Two types of active cartilage are observed: one type is local and self-limiting and usually does not obliterate the fibrous fissula; the other is more vigorously proliferating and may obliterate not only the fissular cleft but extend into the surrounding marrow spaces and impinge upon intervening bony trabeculae. In all cases observed, the latter type of cartilage is wholly different from the mature cartilage surrounding the fissular tract, since it shows evidence of marked cellular activity or tinctorial changes in the matrix characteristic of a stage preparatory to ossification. Later, in the advanced fetus or in the infant, either areas of newly formed gnarly bone may be found, or resorption and rebuilding of bone in the fissular area may occur.

Since the fissula ante fenestram in some specimens of developing otic capsules is capacious beyond regular dimensions, it appears that the invasive periotic tissue, during the process of formation, passes beyond ordinary bounds to produce a large tract. When histologic maturation of this relatively extensive zone of elementary connective tissue is effected, the production of cartilage serves to obliterate part or all of the fissular contents.

These observations permit a correlation between the occurrence of large fissulae and the formation of cartilage masses and suggest a reasonable explanation of the frequent occurrence of sclerotic bone in the antefenestral area. Assumedly, in the latter instance, the cartilage is not slowly converted into intrachondral and endochondral bone but is displaced by a vascular, gnarly bone whose growth occurs at the expense of original capsular architecture. The ultimate formation of sclerotic bone would seem to represent the next stage in

the same continuous process of histologic maturation and replacement. Were this new bone to spread by exuberant growth beyond its site of origin, it may come to involve anatomically important elements in the auditory apparatus; impingement upon, and immobilization of, the stapes may then be manifested as clinical otosclerosis.

SUMMARY

The report of a study of the fissular region of the human otic capsule, based upon an examination of 300 series of sections, is herein concluded.

The range of histologic change within the fissular region has been determined throughout the normal course of fetal development, through the phase of rapid rebuilding during early postnatal years, and through that of slower structural modification in adulthood. Additionally, aberrant types of fissulae have been considered, especially in relation to the presence of chondromatous masses. Observations suggest that the presence of newly formed cartilage is causally linked with the occurrence of exceptionally capacious fissular channels, and the formation of abnormal bone to the antecedent appearance of the cartilaginous nodules.

For further study of the latter step in what appears to be a histologic succession, additional material is required and is currently being assembled in the otological collections at Wisconsin and Northwestern. Only when the genesis of each participant tissue is further clarified and the degree of their interdependence better established, can more definite conclusions be reached.

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REFERENCES

1. Alexander, G.: Das gehörorgan der Kretinen, Arch. f. Ohrenh. 78:54-128, 1909.

2. Anson, B. J., and Wilson, J. G.: The Fissula ante Fenestram in an Adult Human Ear, Anat. Rec. 56:383-393, 1933.

3. Anson, B. J., and Martin, J.: Fissula Ante Fenestram; Its Form and Contents in Early Life, Arch. Otolaryng. 21:303-323, 1935.

4. Anson, B. J., Cauldwell, E. W., and Bast, T. H.: The Fissula Ante Fenestram of the Human Otic Capsule: I. Developmental and Normal Adult Structure, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 56:957-985, 1947.

5. Bast, T. H.: Ossification of the Otic Capsule in Human Fetuses, Contrib. Embryol. Carnegie Inst. (No. 121) 21:53-82.

6. Bast, T. H.: Development of the Otic Capsule: II. The Origin, Development, and Significance of the Fissula Ante Fenestram, Arch. Otolaryng. 18:1-20, 1933.

7. Bast, T. H.: Development of the Otic Capsule: III. Fetal and Infantile Changes in the Fissular Region, Arch. Otolaryng. 23:509-525, 1936.

8. Bast, T. H.: Development of the Otic Capsule: V. Residual Cartilages and Defective Ossification and Their Relation to Otosclerotic Foci, Arch. Otolaryng. 32:771-782, 1940.

9. Bruhl, G.: Zwei Falle von Stapesankylose mit Beteiligung des Hornerven, davon einer im neber diagnostiziert, Ztschr. f. Ohrenh. 50:274-281, 1905.

10. Bruhl, G.: Mikroskopische Demonstration zur Otosklerose, (Discussion of Politzer's paper), Verhandl. d. Deutsch. otol. Gesellsch. 17:113, 1908.

11. Bruhl, G.: Histologische Untersuchung der Felsenbeins eines zu Lebzeiten diagnostizierten Falles von beiderseitiger knocherner Stapesankylose (Otosklerose), Monatschr. f. Ohrenh. 46:1289-1313, 1912.

12. Brunner, H.: Beitrage zur Histologenese des otosklerotischen Knochens, Ztschr. f. Hals-, Nasen-u. Ohrenh. 6:320-334, 1923.

13. Covell, W. P.: The Ossicles in Otosclerosis, Acta oto-laryng. 28:263-276, 1940.

14. Doderlein: Pathologisch-Anatomische Untersuchungen uber die Otosklerose, Ztschr. f. Hals-, Nasen-u. Ohrenh. 6:477-498, 1923.

15. Eckert, A.: Beitrage zum Otosklerose-und Stauungsproblem, Ztschr. f. Hals-, Nasen-u. Ohrenh. 2:449-484, 1922.

16. Fischer, J.: Zur Frage der konstitutionell-kongenitalen charakters des Otosklerose. Morphologische Anomalien des Innenohres als Ausdruck der konstitutionellen Minderwertigkeit des Gehororgans, Monatschr. f. Ohrenh. 55:31-35; 127-143, 1921.

17. Grunberg, K.: Zur Pathogenese der Otosklerose, Ztschr. f. Laryng., Rhin. u. i. Grenzgebiete. 15:78, 1926.

18. Guild, S. R.: Histologic Otosclerosis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 53:246-266, 1944.

19. Habermann, J.: Zur Pathologie der Taubstummheit und der Fensternichen, Arch. f. Ohrenh. 53:52-67, 1901.

20. Habermann, J.: Zur Pathologie der sogenannten Otosklerose, Arch. f. Ohrenh. 60:37-96, 1903.

21. Hagens, E. H.: Otosclerosis, Arch. Otolaryng. 12:273-289, 1930.

22. Hartmann, E.: Zwei neue Falle von doppelseitige knocherner Stapesankylose, Ztschr. f. Ohrenh 33:103-151, 1898.

23. Katz, L.: Sogenannte Otosklerose bei der Katze, Arch. f. Ohrenh. 68:122-134, 1906.

24. Kelemen, G.: Otosclerotic Focus Outside the Inner Ear Capsule, Laryngoscope 53:528-532, 1943.

25. Kosokabe, H.: Ueber die Knorpelfugen in der Labyrinthkapsel bein Embryo, Kinde und Erwachsenen und uber deren Zusammenhang mit der Entstehung der otosklerotischen (otospongiotischen) Herdes, Stuttgart, A. Kernen, 1922.

26. Lange, W.: Ueber die Morphologie und Genese der otosklerotischen Knochenerkrankung, Beitr. z. Anat. 16:189-213, 1921.

27. Manasse, P.: Demonstration mikroskopischer Praparate von sogenannter Stapesankylose, Verhandl. d. Deutsch. otol. Gesellsch. 9:117-118, 1900.

28. Manasse, P.: Ueber die sogenannte Otosklerose, Verhandl. d. Deutsch. otol. Gesellsch. 18:234-241, 1909.

29. Manasse, P.: Handbuch der pathologischen Anatomie des menschlichen Ohres, Wiesbaden, J. F. Bergmann, 1917, pp. 211-220.

30. Mayer, O.: Zur Pathogenese und Aetiologie der Otosklerose, Monatschr. f. Ohrenh. 45:421-437, 1911.

31. Mayer, O.: Untersuchungen uber die Otosklerose, Wien, A. Hoelder, 1917.

32. Mayer, O.: Bericht uber die Ergebnisse weiterer Untersuchungen zur Otosklerosefrage, Ztschr. f. Hals-, Nasen-u. Ohrenh. 6:280-319, 1923.

33. Moller, J.: Quelques remarques sur l'otosclerose a propos d'un cas d'autopsie, Ann. d. mal. de l'oreille 31:235-240, 1904.

34. Nager, F. R.: In: Denker and Kahler, Handbuch d. Hals-Nasen-Ohrenheilkunde, Berlin, J. Springer, 1926, vol. 6, pp. 636-654.

35. Panse, R.: Die Diagnose Otosklerose ist augzugeben, Verhandl. d. Deutsch. otol. Gesellsch. 20:257-267, 1911.

36. Politzer, A.: Ueber primare Erkrankung der knocherner Labyrinthkapsel, Ztschr. f. Ohrenh. 25:309-327, 1893.

37. Ruttin, E.: Osteopsathyrose und Otosklerose, Ztschr. f. Hals-, Nasen-u. Ohrenh. 3:263-279, 1922.

38. Shambaugh, G. E., Jr., and Cutler, M. H.: Progress in Otolaryngology: Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Inner Ear, Arch. Otolaryng. 41:147, 1945.

39. Schilling, R.: Ein Fall von klinisch und anatomisch Untersuchter Stapesankylose, Arch. f. Ohrenh. 68:209-222, 1906.

40. Siebenmann, F.: Multiple Spongiosierung der Labyrinthkapsel als Sectionsbefund bei einem Fall von progessiver Schwerhorigkeit, Ztschr. f. Ohrenh. 34:356-374, 1899.

41. Siebenmann, F.: Totaler knocherner Verschluss beider Labyrinthfenster und Labyrinthitis serosa infolge progressiver Spongiosierung, Verhandl. d. Deutsch. otol. Gesellsch. 20:267-269, 1911.

42. Wilson, J. G.: Fissula Ante Fenestram and Adjacent Tissues in the Human Otic Capsule, Acta oto-laryng. 22:382-389, 1935.

43. Wittmaack, K.: Ueber einen Befund von wahrer Spongiosierung der Labyrinthkapsel, Ztschr. f. Ohrenh. 77:201-206, 1918.

44. Wittmaack, K.: Die Otosklerose auf Grund einiger Forschungen, Jena, G. Fischer, 1919.

45. Wittmaack, K.: Ein bemerksenwerter Fall von Otosklerose, Der Hals-, Nasen-u. Ohrenarzt. 32:1-24, 1941.

46. Wolff, H. I.: Ein Beitrag zur Pathologie der Otosklerose (Ostitis chronica metaplastica), Beitr. z. Anat. 7:274-295, 1914.

VIII

BRONCHOSCOPIC EXPERIENCES WITH TUMORS OF THE LOWER RESPIRATORY TRACT

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The diagnosis and treatment of tumors of the lower respiratory tract is a splendid example of the close relationship between many different branches of medicine and surgery.

That there are many cases of tumors of the lungs and the trachea is well realized today, but a correct diagnosis may require repeated examinations. These tumors are found five times as frequently in males as in females. They occur most often between the fourth and sixth decades. Malignant tumors are far more common than benign tumors, and two thirds of the malignant tumors are squamous cell carcinoma, which are relatively slow growing and metastasize late. A benign tumor may from its situation be as deadly as a malignant tumor.

The symptoms of fever, cough, pain and hemoptysis are present in the above order. These are signs of either bronchial irritation or obstruction.

X-ray examination of the chest is a most important preliminary diagnostic procedure. On the other hand, in a small percentage of cases, x-ray examination may fail to reveal a tumor. In cases in which malignant pulmonic disease is suspected, lateral as well as anterior-posterior views should be taken routinely.

When a tumor occludes a large bronchus and lipiodol is placed in the bronchus, a characteristic x-ray picture is shown; namely, a pool of lipiodol situated at the site of the lesion. Pulmonary hemorrhages are common symptoms of a new growth of the lung, and because pulmonary hemorrhage is so closely allied with pulmonary tuberculosis, many of these unfortunate people are considered tubercular and are treated in tuberculosis sanatoriums. In any patient with hemoptysis and negative sputum findings for tuberculosis bacilli,

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further steps must be taken for a complete diagnosis. In many cases of pulmonary tumors in which there is hemoptysis, the sputum is not the bright red, frothy sputum of the textbooks, but rather dark clots that are coughed up at intervals by the patient. Also a vague feeling of pain in the chest has been a noticeable symptom. An infection, which is blocked in by a new growth, may overshadow the underlying cause of the malady entirely.

A bronchoscopic examination affords many important facts leading towards a diagnosis: the color of the mucous membrane of the trachea and bronchi; the size, position and shape of the lumina of the trachea and the bronchi; the contour of the carina; the fixation of the primary bronchi; the orifices of the upper lobe bronchi, which can only be inspected satisfactorily by the use of a telescope; and the status of the right middle lobe bronchus and the major divisions of the lower lobe bronchi on each side. When a growth is situated in a large bronchus of the lower lobe or is near the orifice of the middle lobe bronchus, tissue can be removed from suspicious areas for microscopical diagnosis. When a suspicious area cannot be visualized through the bronchoscope, mucus or washings from this area may, when studied, show malignant cells which have broken away from the tumor mass and have been carried by mucus into the larger bronchi. The application of Papanicolau's[7] technique, as developed by Clerf,[8] has been of great aid in the early positive diagnosis of malignant tumors beyond the view of the bronchoscopist.

The following are five cases that illustrate important points in the diagnosis of lung tumors:

CASE 1.—A white male, aged 40, was admitted to the dispensary of the Johns Hopkins Hospital, complaining of increasing dyspnea on exertion of one year's duration. The family history and the past history were negative. He had lately noticed on exertion a wheezing noise in his throat, also a slight cough. Physical examination was negative except for an expiratory wheeze and a decrease of breath sounds in the left upper chest. The bronchoscopic examination disclosed a large growth, which bled easily, arising from the left wall of the trachea just above the bifurcation. The section showed squamous cell carcinoma. The tumor was thoroughly fulgurated. The patient had a stormy time for the next 24 hours, developing a massive collapse of the left lung. However, this cleared up promptly and the patient was apparently well. He gained 30 pounds, and came for fulguration of the tumor at regular intervals. He lived in comparative comfort for 10½ years, though finally succumbed to the malignancy 11 years after diagnosis.

CASE 2.—A white female, aged 24, was admitted to the Johns Hopkins Hospital because of frequent and sudden hemoptysis and a dull pain below the left breast, which was fairly severe at times. The lungs were clear to percussion and auscultation. X-ray examination of the chest was negative. The bronchoscopic examination disclosed a small tumor just at or slightly below the bifurcation of the left bronchus. A section of tissue was reported finally as an invasive cylindroma. Many attempts were made to reduce this tumor by biting forceps, fulguration and radiation without success, and the patient was referred to a surgeon for operation. The left lung was excised; the patient made a complete recovery and has remained well. Hemoptysis is frequently a symptom of a bronchial tumor, especially adenoma or cylindroma. Also atelectasis of part of the lung may be present but be hidden behind the heart shadow and fail to show in the x-ray films.

CASE 3.—A white child, female, aged 3, was admitted to the Harriet Lane Home for diagnosis. The past history was negative until April 1933 when she began to vomit. An examination by her family physician disclosed pronounced dullness over the left chest, with marked bronchial breathing. She entered the Harriet Lane Home with the same findings. X-ray examination disclosed displacement of the heart and the lungs to the left side, with the center of the left lung hazy. The bronchoscopic examination revealed a glistening, yellowish mass filling the left bronchus and extending into the trachea. A section of this was first diagnosed as fibrosarcoma, later as fibroma. Lipiodol injected into the trachea failed to enter the left bronchus, although the child was turned on her left side. An attempt was made to remove the tumor through the bronchoscope but this was impossible. It was then decided that an exploratory operation should be done for the probable removal of the lung. The left lung was removed and the child remained well, but was drowned while on a picnic three years later. Atelectasis is caused by the occlusion of the bronchi. Atelectasis of the whole lung is caused by occlusion of a primary bronchus.

CASE 4.—A white female, aged 30, entered the Union Memorial Hospital, December 1933, with hemoptysis. The patient was first admitted to the hospital four years before with fever and cough. Physical examination disclosed fullness over the left upper chest, with distant breath sounds and limited diaphragmatic descent on the left. The x-ray diagnosis was tuberculosis, left upper chest. A few acid bacilli were seen in the sputum. She was sent to a tuberculosis sanatorium. In March 1932, she had hemoptysis after an artificial pneumothorax. Later, she had a thoracoplasty, and later

still a phrenecostomy. After her last operation, she improved greatly in general health, gained weight, but the hemoptysis continued. Endoscopic examination showed the left bronchus completely occluded by a growth, which bled easily. A section of this was diagnosed as cylindroma of the bronchus. The fact that a patient has tuberculosis does not exclude the possibility of his also having a lung tumor.

CASE 5.—A white male, aged 48, entered the Johns Hopkins Hospital June 1935, with pain in the chest and fever from an unknown cause. The present illness began in 1934 with a feeling of fatigue. Complete and thorough examination at that time, together with x-ray films of the chest, was entirely negative, and the patient was told that he was merely suffering from nervousness. Shortly following this, he developed what was diagnosed as influenza, and at that time had a cough along with the pain in his chest. Bronchoscopic examination in June 1935 showed purulent discharge in the right main bronchus, and that the bronchus was occluded from without. The patient was operated on and several lung abscesses were opened and drained. The wound did not heal, however. The patient continued to feel bad and was sent again in October for bronchoscopy. At this time, the right bronchus was occluded by a growth which bled easily. Microscopic examination of a section showed squamous cell carcinoma. This case illustrates two points: that an infection may completely overshadow a new growth; that careful, repeated examination may be necessary to arrive at a correct diagnosis.

Bronchoscopic extirpation of carcinoma offers little or no hope for cure. Benign tumors on the other hand may be successfully removed.

When a diagnosis of carcinoma of the lung has been made, the question of lobectomy or pneumonectomy should be given consideration at once, and in our experience only surgical removal of the entire carcinoma, including carcinomatous glands, effects a cure.

SUMMARY

1. Dyspnea may be due to partial occlusion of the trachea. A laminagraph may help in determining the extent of the growth.

2. Frequent hemoptysis beginning and ending suddenly is a common symptom of a bronchial tumor, frequently a cylindroma or an adenoma.

3. Atelectasis of an entire lung or any part of it is caused by occlusion of a bronchus. Only the upper portion of a tumor is seen through the bronchoscope if the lumen of the bronchus is filled.

4. A positive sputum test for tubercle bacilli or a positive Wassermann test does not exclude the possibility of other diseases of the lung.

5. Lipiodol, when placed in a bronchus occluded by a tumor, forms a characteristic pool and x-ray shadow at the site of the lesion.

6. A secondary infection may completely obscure an underlying new growth.

7. Cytological study of bronchial mucus, as developed by Clerf, has aided in early diagnosis of pulmonic malignancy.

8. An endoscopic examination can be the solution of a most complicated pulmonary picture.

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REFERENCES

1. Clerf, L. H., and Crawford, B. L.: Bronchogenic Carcinoma, J. Thoracic Surg. 3:73, 1933.

2. Samson, P. C.: Entdifferentiation in Bronchogenic Carcinoma, Am. J. Cancer 23:741, 1935.

3. Jackson, C.: Endothelioma of the Right Bronchus Removed by Peroral Bronchoscopy, Am. J. M. Sc. 153:371, 1917.

4. Graham, E. A., and Singer, J. J.: Successful Removal of an Entire Lung for Carcinoma of the Bronchus, J. A. M. A. 101:1371, 1933.

5. Rienhoff, W. F., Jr.: Graded Pneumonectomy in the Treatment of Tumors of the Lung, Bull. Johns Hopkins Hosp. 64:167, 1939.

6. Broyles, E. N., and Fisher, G. E.: Bronchoscopic Experiences with Lung Tumors, Surg. 7:918-923 (June) 1940.

7. Papanicolaou, G. N., and Olcott, C. T.: Studies of Spontaneous Tumors in Guinea Pigs; Tumors of Stomach and Intestine, Arch. Path. 34:218-228 (July) 1942.

8. Clerf, L. H., and Herbut, P. A.: Bronchogenic Carcinoma; Diagnosis by Cytologic Study of Bronchoscopically Removed Secretions, J. A. M. A. 130:1006-1012 (Apr. 13) 1946.

9. Ochsner, A., DeBakey, M. E., and Dixon, L.: Primary Pulmonary Malignancy Treated by Resection, Ann. Surg. 125:522 (May) 1947.

IX

TREATMENT OF CHRONIC NASAL DIPHTHERIA CARRIERS: REPORT OF CASE

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In the present paper, the treatment of chronic nasal diphtheria carriers will be reviewed, and the successful treatment of an unusually resistant carrier will be reported. In approximately 70 per cent of cases, cultures of material from persons who have diphtheria will become negative three weeks after the onset of the disease. In the remaining 30 per cent, the patients usually become free of Corynebacterium diphtheriae after variable longer intervals, except for a few persons who become chronic carriers of the disease.

Diphtheria may manifest itself clinically in various locations, including the pharynx, nose, larynx, middle ear, conjunctiva, skin and genitalia. Education of the public, combined with the requirement of successful immunization against diphtheria prior to children's entering school, has done much to make clinical diphtheria a rarity.

The most common variety of clinical diphtheria by far is the pharyngeal type. As a result, insignificant notice is accorded the relatively rarer types of diphtheria. Nevertheless, it should be remembered that the disease can occur at the aforementioned various locations, particularly in the nose. In addition, subclinical forms of diphtheria undoubtedly occur. This fact is borne out by the accidental discovery of virulent diphtheria organisms in both patients and employees in institutions in which cultures are made routinely. After this discovery, the careful taking of a history in the majority of cases reveals few or no significant symptoms.

The finding of a fetid, irritating unilateral or bilateral nasal discharge causing excoriation and swelling of the alae nasi and the

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upper lip should arouse suspicion of the presence of diphtheria organisms. When patients have a chronic nasal discharge, the routine submission of specimens for culture will, on occasion, reveal Corynebacterium diphtheriae among the etiologic organisms.

Since all strains of Corynebacterium diphtheriae are not pathogenic, it is necessary that a test of virulence be made to determine the status of cultured organisms. Moreover, there is considerable evidence that a patient may acquire a virulent strain of Corynebacterium diphtheriae and then carry it in the nose indefinitely without any clinical manifestations of toxic effects. The protection afforded in these cases is ascribed to the process of auto-immunization as shown by De Stella.[1] The virulence of the organisms lodging in the nasal mucosa may become attenuated so that the organisms are harmless. At some future period, the virulence of these organisms may be reactivated by either local trauma or an infection with other bacterial forms. There are numerous reports of cases which seem to bear out this belief. For example, Wilner[2] reported three cases in which virulent diphtheria organisms were found after the removal of foreign bodies from noses which, when previously examined, had appeared to be normal. Similarly, O'Reilly[3] reported a case in which a boy, nine years old, had sustained a severe injury to the bridge of the nose one week before nasal diphtheria developed.

In such cases, local nasal trauma apparently served as the activating factor for the virulence of previously existing and almost harmless diphtheria organisms. Costen[4] reported on a child from whose discharge nonvirulent strains of Corynebacterium diphtheriae were obtained by culture after a respiratory infection. In this instance, the respiratory infection appeared to be the factor in the activation of the virulence of the diphtheria organisms. It would seem advisable to observe carriers of nonvirulent diphtheria organisms and to redetermine the virulence of the organisms from time to time, especially after such persons have had infections of the upper part of the respiratory tract or any nasal trauma.

A review of the literature reveals that a variety of therapeutic measures have been and still are being utilized in the treatment of diphtheria carriers. On the whole, the reported results are surprisingly good. In 1913, McCollum and Place[5] reported that no satisfactory means had been found to cure carriers of the disease.

The use of antiseptic nose and throat sprays and the painting of throats with solutions of iodine or formaldehyde have not been satisfactory. Diphtheria antitoxins also have been employed in the

treatment of carriers with little success. Failure of such antitoxins is attributed to the fact that they exert no direct action on the organisms themselves.

Tonsillectomy and adenoidectomy, combined with spraying of the nose and throat with antiseptic or chemotherapeutic agents, frequently have proved successful.

The employment of roentgen therapy in the treatment of chronic diphtheria carriers is more widely resorted to in Europe than in America. Roentgenologists report a high percentage of cases in which diphtheria carriers are treated with roentgen rays. The action of the roentgen rays is not directly bactericidal, as has been proved experimentally. Kaplan,[6] however, said that experience has shown that irradiation affects inflammatory conditions favorably. The endotoxins that are formed are said to destroy the bacteria. In addition, fibrosis later occurs, with shrinkage of tonsillar tissue and elimination of the formation of crypts which are so favorable to the lodgment of the diphtheria organisms. The experimental work of Heineke[7] and that of Warthin[8] showed that lymphocytes disintegrate as soon as fifteen minutes after roentgen therapy and that this continues for several days. Desjardins[9] and Hodges and Berger[10] associated this destruction of lymphocytes with the liberation of large quantities of active antibodies which are bactericidal. In 1922 Hickey[11] reported his results in the roentgen treatment of a group of carriers of diphtheria. In his group, 19 persons carried the organisms in the throat and six carried them in the nose; in nine the organisms were associated with otitis media. Hickey reported cures in 15 of the persons who carried the organisms in the throat, in four of those who carried the organisms in the nose, and in four of those who had otitis media.

Kaplan reported treatment of a total of 11 chronic diphtheria carriers of various types. Cures were obtained in all cases by means of high voltage roentgen rays. He found that three treatments at weekly intervals were adequate to achieve the desired results. Kaplan also reviewed the successful results of other roentgenologists. Particularly worth noting, in Kaplan's review, are Streil's[12] summarized results in the roentgen treatment of 150 diphtheritic persons. Streil's series consisted of two groups. In the first group were those persons who had recovered from the clinical form of diphtheria, but from whom smears containing diphtheria organisms still were obtained three or four weeks after the onset of the disease. The second group consisted of diphtheria carriers who had been discovered accidentally. Streil was said to have achieved good results for 95 per cent of per-

sons in the first group and for 97 per cent of persons in the second group. This form of roentgen therapy is said to be harmless, and it appears to be highly effective.

The use of chemotherapeutic agents in the treatment of chronic diphtheria carriers seems generally to be very favorable. In London, Boissard and Fry[13] treated 26 children who were chronic nasal diphtheria carriers and who were also found to have nasal infections caused by hemolytic forms of streptococci. In their series, the intranasal insufflation of sulfanilamide was carried out morning and evening for eight days. After the first course, the nasal infection was eradicated completely from 24 of the patients. The remaining two patients were freed of infection after a second course. Boissard and Fry concluded that the persistence of diphtheria organisms in the nose depends mainly on the question of whether the associated hemolytic streptococcic infection will or will not respond well to the insufflation of sulfanilamide powder.

Legros[14] used aerosols of sulfapyridine with success in the treatment of diphtheria carriers. Skinner[15] found that the intramuscular injection of penicillin was effective in the treatment of 20 persons in whom culture of material from the throat repeatedly produced virulent organisms. Total daily doses of 50,000 to 100,000 units of penicillin were administered, and an average of 16 days was required to obtain cures. Every patient in his series was benefited.

Thrift[16] reported the successful treatment of a persistent nasal form of diphtheria with penicillin. On the other hand, Herrell[17] found the effectiveness of penicillin disappointing in the treatment of diphtheria carriers. Despite the intramuscular administration of large amounts of penicillin for one week to two weeks, results of culture were negative in only a few cases during the time penicillin was administered or shortly after the course had been completed. Herrell said that there were recurrences among those few patients cultures of whose secretions had become negative. He wrote that if penicillin is to be used against the infection, he would without exception recommend that administration of it be combined with standard procedures, such as the use of diphtheria antitoxin.

In the case to be reported herein, various forms of local therapy proved ineffective over a two-year period prior to the patient's coming to the clinic.

REPORT OF A CASE

A white man, 71 years old, was seen in the Section on Otolaryngology and Rhinology on June 18, 1947. He complained that: "I've

got a bad throat. I'm a diphtheria carrier." He had been found to be a carrier of this disease in March 1945, after routine culture of material from the nose and throat at a state hospital in which he was employed. Signs and symptoms included a purulent nasal discharge and variable degrees of irritation of the throat for a few years prior to his coming to the clinic. He was known to have had bronchial asthma for about 25 years. The patient never had been immunized against diphtheria.

At the age of seven years the patient had had diphtheria. His tonsils had been removed in 1919. Nasal polyps had been removed, first in 1924 and seven times subsequently. Measures adopted by the patient to alter his status as a carrier of diphtheria included the persistent local application of various antiseptic agents and sprays for two years, without success.

At physical examination pus was found in the right side of the nose, the turbinate bones and the nasal septum were almost entirely absent, crusting and atrophy of the membranes were marked, the tonsils had been removed cleanly and the mucosa of the pharynx was mildly granular. The patient was found to have hypertensive cardiovascular disease.

Roentgenograms disclosed diffuse opacity of all sinuses (Figs. 1 and 2*a* and *b*). Results of culture of material from the nose and throat taken on June 18, July 2 and July 9, 1947, were positive, and the diphtheria organisms thus produced were virulent. Results of routine studies of the blood were normal.

Because of the chronic sinusitis and the past resistance of the patient's state to conservative therapy, intranasal surgical treatment combined with the use of penicillin and diphtheria antitoxin was instituted. The patient was hospitalized on July 28, 1947. A three-day preoperative course of penicillin administered intramuscularly and by nasal atomizer was carried out. The patient received 40,000 units every three hours intramuscularly and an additional 40,000 units every two hours by nasal atomizer during his waking hours. Operation was performed on July 31; bilateral intranasal windows were made. The lining membranes were found to be thickened and pus was present in the right antrum. Normal mucous membrane was not found anywhere in the nose. Masses of fungating granulation tissue on the floor of the nose extending posteriorly were removed carefully. Although there was bleeding to some extent, epinephrine was not used. The patient's nose was packed with petrolatum gauze. Penicillin was given intramuscularly throughout the patient's stay

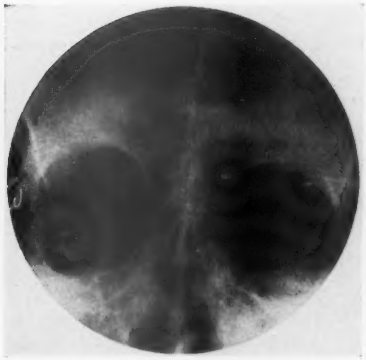

Fig. 1.—Some evidence of reactive osteitis in frontal sinuses.

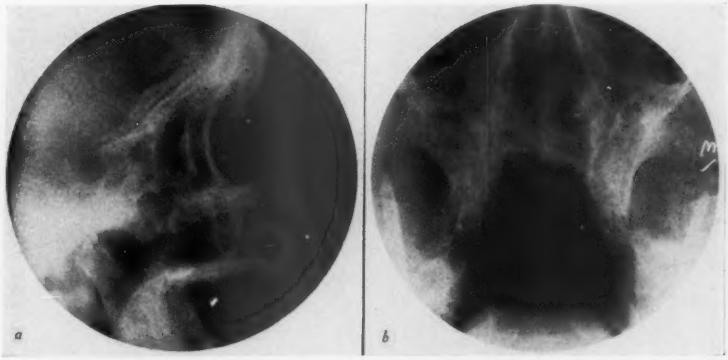

Fig. 2.—Diffuse clouding of all the sinuses. *a*. Lateral view; *b*, view of maxillary sinuses.

in the hospital. He was given 20,000 units of diphtheria antitoxin a few hours following his return to his room after operation. Convalescence was uneventful and the patient was dismissed on the fifth postoperative day.

Four successive cultures of material from the nose and throat were submitted during a period of six weeks and all were negative for Corynebacterium diphtheriae. The patient returned for another examination and the last culture was made on November 28, 1947. Results were reported as negative (Table 1).

COMMENT AND SUMMARY

Roentgen rays, sulfonamides and penicillin are usually effective therapeutic agents in the treatment of chronic nasal diphtheria carriers. However, the presence of chronic sinusitis in a nasal diphtheria carrier is an indication for surgical procedures combined with chemotherapy if a cure is to be obtained.

Despite the infrequency of the occurrence of diphtheria organisms among nonimmunized patients who have chronic nasal infections, material from the nose should be cultured for diphtheria organisms.

Carriers who have been cured should be checked periodically to make certain that the organisms have not reappeared, especially following a severe upper respiratory infection or nasal trauma.

MAYO CLINIC.

TABLE 1.

RECORD OF CULTURES FROM NOSE AND THROAT.

DATES	CULTURES NUMBER	RESULTS*
Cultures at State Laboratory		
1945		
March 22 through May 9	27	Positive
May 10	1	Negative
May 15 and 16	2	Positive
May 17 through 19	3	Negative
1947		
May 8	1	Reserved
May 13 through July 10	8	Positive
Cultures at Mayo Clinic		
June 18 through July 9 (Penicillin July 28 to August 5; operation July 31)	3	Positive
August 4 through 19	3	Negative
Cultures at State Laboratory		
September 12	1	Negative

*Results of virulence test positive March 28, 1945, May 14, June 18, and July 2, 1947.

REFERENCES

1. De Stella, H.: La rhinite diphtérique expérimentale, Arch. internat. de laryngol. Par. 16:970-974, 1903.

2. Wilner, Anna S.: Primary Nasal Diphtheria; A Plea for Its Early Recognition and a Report of Three Cases, New York M. J. 80:881-883 (Nov. 5) 1904.

3. O'Reilly, W. F.: A Case of Nasal Diphtheria with Misleading History, J. A. M. A. 82:631 (Feb. 23) 1924.

4. Costen, J. B.: Nasal Diphtheria; Report of Four Cases, One with Rhinolith, J. Missouri M. A. 36:266-268 (July) 1939.

5. McCollom, J. H., and Place, E. H.: Diphtheria. In Osler, William, and McCrae, Thomas: Modern Medicine; Its Theory and Practice, Philadelphia, Lea & Febiger, 1913, chapt. 18, p. 732.

6. Kaplan, I. I.: Radiation Therapy of Diphtheria Carriers, Radiology 35:425-428 (Oct.) 1940.

7. Heineke, H.: Ueber die Einwirkung der Röntgenstrahlen auf Tiere, München. med. Wchnschr. 50:2090-2092 (Dec.) 1903.

8. Warthin, A. S.: An Experimental Study of the Effects of Roentgen Rays upon the Blood-Forming Organs, with Special References to the Treatment of Leukemia, Internat. Clin. 4:243-277, 1906.

9. Desjardins, A. U.: Radiotherapy for Inflammatory Conditions, J. A. M. A. 96:401-407 (Feb. 7) 1931.

10. Hodges, F. M., and Berger, R. A.: Roentgen Therapy of Some Infections, J. A. M. A. 107:1551-1554 (Nov. 7) 1936.

11. Hickey, P. M.: Treatment of Diphtheria Carriers by Means of the Roentgen Rays, Am. J. Roentgenol. 9:319-321 (May) 1922.

12. Streil, W.: Quoted by Kaplan, I. I.[6]

13. Boissard, Joan M., and Fry, R. M.: Chronic Nasal Diphtheria Carriers; Cure with Sulphanilamide, Lancet. 1:610-614 (May 23) 1942.

14. Legros, J.: Essai de traitement des porteurs de germes diphtériques par aérosols de sulfapyridine, Presse méd. 51:332 (June 19) 1943; (Abstr.) Treatment of Diphtheria Carriers with Aerosols of Sulphapyridine, Bull. Hyg. 11:893 (Nov.) 1943.

15. Skinner, J. W.: Penicillin in Persistent Diphtheria; Treatment of Patients with Repeated Positive Throat Cultures, U. S. Nav. M. Bull. 45:264-266 (Aug.) 1945.

16. Thrift, G. N.: The Report of a Persistent Nasal Diphtheria Carrier Treated with Penicillin, M. Bull. Mediterranean Theat. Op. 3:19-20 (Jan.) 1945.

17. Herrell, W. E.: Penicillin and Other Antibiotic Agents, Philadelphia, W. B. Saunders Company, 1945, p. 150.

X

THE REPAIR OF THE EAR DRUM IN BLAST INJURIES

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Rupture of the ear drum was the most common early aural lesion encountered in blast injuries in World War II.[1, 6, 19] It has been also the most common aural injury in peacetime.

There are some differences in the characteristics of an injury sustained in wartime and one sustained in peacetime, namely, the location of the perforation and the incidence of infection. The basic reparative processes of the drumhead in both are, however, the same, so that in the management of peacetime perforations, we should take cognizance of the extensive experience gained in the observation of the war injuries.

Types of blast[9] have been classified as: atmospheric (from detonation of aerial bombs, large caliber shells, mortar shells, hand grenades, land mines), and immersion blast (from detonation of depth charges in water). Air-borne pressures are considerably less than water-borne pressures.

Anyone within the critical distance from the center of these blasts is subject to severe damage to his auditory apparatus. The critical distance in air is said to be 20 feet; that in water, about 80 feet.[17]

There are two phases[5, 9] to the atmospheric blast: a powerful and shorter pressure component, and a weaker and longer suction component.

There are also two phases[9] to the immersion blast: the "a" phase, or compression wave, which is the destructive factor, and the "b" phase, the disturbance produced by the expanding bubble of gases liberated by the explosion. The "b" phase is not destructive.

The degree of damage done to the ear drum ranges from a small interstitial hemorrhage to almost total destruction.[5]

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Observers[3, 5, 7, 10, 19] generally agree that rupture of the tympanic membrane seems to exert a protective influence on the labyrinth and cochlea and that the degree of deafness produced is greater when the drum is not ruptured. This has been confirmed experimentally by Guild.[10]

Some theories as to the mechanism of rupture of the drum are: (1) the intense detonation waves compress the air in the external auditory canal to several times the normal atmospheric pressure and rupture the drum;[10] (2) the drum possibly withstands the compressive force and then ruptures *outward* by suction acting suddenly in the opposite direction for a longer period[5] (The eversion of the edges present in some perforations is thought by certain observers to be produced by the wave of suction); (3) the pathway of the blast is not through the external auditory canal, but through the eustachian tube. This could account for the high incidence of anterior perforations.[4]

Various observers have pointed out certain features in blast rupture of the ear drum.

Deafness was of the middle ear type[18] superimposed upon a temporary perception deafness. The subsequent damage to hearing in most cases was not marked.

Early tinnitus, which generally disappeared, was invariably present.

The incidence of infection was higher in war than in peacetime. Alexander[1] stated that in 50 per cent of the cases of perforations in wartime there was infection and suppuration as compared with 20 per cent of those of peacetime. In Silcox and Schenck's[19] series, infection occurred in 42.6 per cent; in Palmer's,[18] 38 per cent; in Collins',[6] 22 per cent; and in Henry's,[11] 17 per cent. In all these series, there were notably few cases which required mastoidectomy.

In contrast to peacetime perforations which favor no definite part of the ear drum, the location of the perforation in the majority of cases was in the anterior inferior quadrant,[2, 18] except in Silcox and Schenck's series in which most were central. Perforation in the attic region was never observed.[5, 7]

It was thought by some[2, 19] that infection diminished the incidence of spontaneous closure of perforations.

In Palmer's[18] series of 100 perforations, 45 per cent healed in from 21 to 28 days. Korkis[14] reported that 57 per cent of 211 per-

forations healed in five weeks. In all the series reported, statistics on the eventual fate of the perforations were not possible because of the frequent transfer of patients.

There was some difference of opinion as to the early management of ruptured ear drums. Alexander[1, 2] and Palmer[18] thought that early insufflation of sulfathiazole or sulfanilamide powder exerted a protection against infection. Silcox and Schenck[19] suggested the use of microcrystalline sulfathiazole.

Craig,[7] Bettington,[3] and Henry[11] were against the use of a sulfonamide powder. Bettington thought that insufflation pushed infected material into the middle ear. Henry felt that the canal wall, drum, and middle ear were slightly more inflamed when a sulfonamide powder was employed.

According to Ireland,[13] at one time in the war the British recommended early insufflation of sulfonamide powder into the ear but later discontinued it as unsuitable and often harmful.

It was generally agreed that the less early manipulation the better. Ear drops and irrigation to remove wax, dirt particles or blood were taboo.

Hoople,[12] Henry,[11] Ireland,[13] and Work[22] used splints or prostheses to increase the incidence of closures. Ireland and Work employed Cargile membrane. Henry used sterile cellophane, which, in addition to enhancing closure, occasionally terminated the tinnitus.

Our service was with the Navy at a South Pacific area where battle casualties came in at varying periods after injury. The number of casualties seen changed with the progress of the island-to-island campaign. They included sea- and land-based Navy and Marine personnel and, during one campaign when casualties were heavy, included an overflow of army personnel. Some casualties were flown in the same day or the day after they were injured; others arrived by ship from four days to several weeks later. "On-the-spot" cases were seen as the result of a catastrophic munitions explosion and, from time to time, after accidental explosions of bombs at nearby camps or from untimely explosions of dynamite caps on maneuvers. This presented a unique arrangement for the study of all types of injury to the auditory apparatus in general and to the ear drums in particular. The opportunity for follow-up depended upon the arrival of ships for casualty evacuation which meant that the patients stayed with us from one to three months or occasionally longer. If

it seemed that the auditory lesion would heal so that the patient could be returned to the line of duty, we were allowed to keep him as long as 120 days. Thus, the ability to predict the outcome became important. In the beginning, we were fooled, prognostically speaking, by the large holes in the ear drums and the amount of infection present, but as time went on, we became better able to predict the lesions which would and which would not be disabling.

Our experiences with blast injuries, with a few exceptions, were in accord with those reported by others. Tinnitus and temporary deafness were constant early symptoms. Transient vertigo, of 8 to 12 hours' duration, was occasionally present. Deafness was of the conduction type superimposed upon a temporary perception deafness. Hearing, in spite of large perforations, was usually good. Hearing was more often impaired in an ear with an unruptured drum. The perforation was located in the posterior and central portions of the drum as frequently as in the anterior portion. A blast perforation of Shrapnell's membrane was not seen in these early cases, although later, at a Stateside hospital, we did see older cases of chronic otitis media and mastoiditis with attic perforations the onset of which was ascribed to a blast injury. Most of these showed a sclerotic type of mastoid on x-ray examination and so were probably predisposed to a chronic infection as suggested by the theory of Wittmaack.[23]

Perforations with infection, contrary to the experience of others,[2] closed spontaneously as often as those without an associated otitis media.

The incidence of infection and suppuration in our series was very high. The exact reasons are not known, but there were some factors that suggested possible causes. Sweating in the humid climate of the South Pacific predisposed to skin infections which were very prevalent. Otitis externa of bacterial and fungus origin was common and possibly infected many of the middle ears through the ruptured drums. Many of the ship casualties were immersed for varying periods of time with the sea water washing infection, foreign particles, and oil into their middle ears through the torn drums.

In the infected middle ears the local use of microcrystalline sulfathiazole did not seem to shorten the course. Sulfadiazine by mouth did appear to influence favorably the more serious cases. Penicillin was not used at that time because it was not available.

Frequent cleansing of the canal and the middle ear (once or twice daily) by topical suction was probably the most effective aid in healing the otitis media.

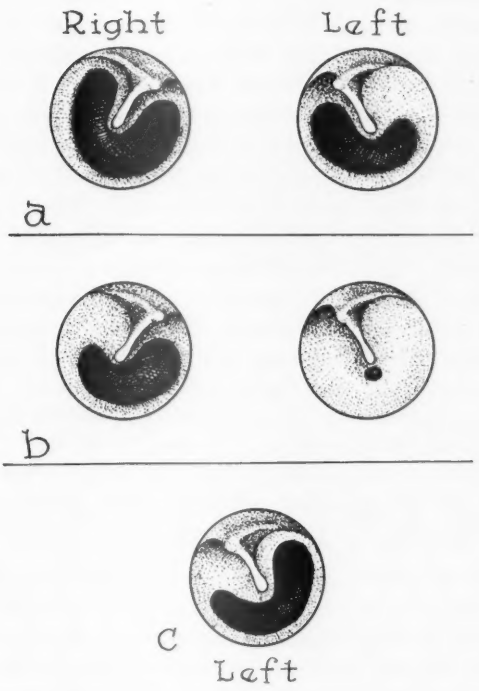

Fig. 1.—Drawings made from on-the-spot diagrams of ear drum membranes; *a*, those of Case 1; *b*, those of Case 2; *c*, that of Case 3.

The most interesting of all observations was the ability of the drum to repair itself even when up to three-fourths of its total area appeared to have been destroyed. To illustrate this the following three cases are reported:

CASE 1.—E. A. K. was seen three days after he had been injured by an explosion of TNT five feet away. In the right ear the perforation involved practically the entire pars tensa. In the left ear the large kidney-shaped perforation was anterior central. Fig. 1*a* is a reproduction of a drawing made when the patient was first seen. It will be noted that there was still a margin of drum remaining near the annulus and the malleus. A slight amount of moisture was present in the right ear. The left was actively discharging. The remaining portions of the drum and the mucous membrane of the

middle ear were very red. Bone conduction was better than air conduction in both ears and the Weber test, using the 512 d.v. fork, was lateralized to the right. Sulfadiazine was given by mouth. None was applied to the ears which were kept clean by daily topical suction. Sixty-three days after the injury, both drums were closed and healed in such a manner as to defy detection of the recent injury. Test by whispered voice at this stage revealed a hearing of 13/15 for the right ear and 15/15 for the left. (An audiometer was not available. Whispered voice proved to be our best means of evaluation of a patient's ability to hear.)

CASE 2.—J. H. J. was injured in the same accident as the previous patient. More than one-half of the anterior portion of the right drum was missing while the left had a small pin-point central perforation (Fig. 1*b*). The right ear was discharging; the left was dry. Bone conduction was greater than air conduction in the right ear. Air conduction was greater than bone in the left. The Weber test was lateralized to the right. The treatment was the same as in Case 1. In only 17 days after the injury both drums were healed. Whispered voice was 15/15 for each ear.

CASE 3.—R. F. J., a newspaper correspondent, was injured when two bombs exploded at distances of 10 and 15 yards. Several hours later he noticed deafness in his left ear with a small amount of blood in the canal. He was first seen ten days after the accident and presented the very large discharging perforation of his left drum noted in Fig. 1*c*. Treatment consisted of daily local cleansing of the ear by topical suction and instillations of microcrystalline sulfathiazole plus sulfadiazine by mouth. Thirty days after the accident the perforation was healed with almost no noticeable scar.

As more and more perforations were seen, it became apparent that size was no factor in the eventual outcome. The location of the perforation was not important but its relation to the tympanic sulcus was, as has recently been pointed out by Hoople[12] and Craig.[7] A margin of drum between the perforation and the sulcus was a requirement for healing. If the perforation extended to the sulcus, closure did not take place. If the malleus retained its drum covering, the chances of repair were enhanced, but a denuded malleus tip did not preclude closure.

The method of drum repair lends itself to interesting speculation. Which of its layers takes the lead in the regenerative process? Does the repair occur concentrically or from an anterior to posterior direction as suggested by Stinson?[21] Stinson believes that the epi-

thelial layer is the active one and has shown by India ink markings on the drum that there is a constant movement of the epithelium from the anterior portion of the drum to the posterior portion, and thence outward along the posterior canal wall.

Dunlap and Schuknecht,[24] who have reported many successful closures of old tympanic perforations with repeated applications of a saturated solution of trichloracetic acid to the margins of the perforations, state that the middle layer of the tympanic membrane is the important one in the healing of perforations by this method. The trichloracetic acid removes the epithelial barrier at the edge of the perforation and, by this tissue destruction, incites a healing response in the margin of the perforation. Fibroblastic proliferation in the middle layer of the granulating margin is responsible for the laying down of interlacing fibers of collagen. When collagen fibers are completely formed they shorten. The shrinking of this circular band of interwoven collagen fibers may facilitate closure by purse-string action.

While this mechanism probably takes place in these chemically stimulated perforations which heal with practically no scar, it does not explain the healing of perforations covered by thin, atrophic drum membrane.

What happens to the drum substance in the large perforations described? Is there a loss of drum tissue or does the drum retract, as suggested by McNaught?[16] The absence of particles of drum in the ear cavity after injury and the rapidity of closure of some traumatic perforations suggest that retraction does occur.

Does infection enhance or impede repair? Alexander[2] considered that a ruptured drum without infection stands a better chance of closing. Hoople,[12] on the other hand, noted that if suppuration followed rupture, it was the rule that with cessation of the discharge, the drums would heal. This, too, was our experience; in fact, it occurred with such regularity that it seemed that large traumatic perforations possibly did better in the presence of infection and that the reaction of inflammation was a stimulus to repair.

Continued infection does, of course, hinder the closure of a drum perforation, but once the infection ceases, a persistent perforation should stir a challenge to close it. During the war, the otologists who used prostheses were rewarded with an increased incidence of healed drums. The same should be true in peacetime.

An artificial membrane to stimulate healing of a perforation is an old device.[8, 20] Cargile membrane, cigarette paper, thin layers of

oiled cotton, rubber tissue, cellophane, fibrin film, and gold foil have been some of the materials used.

Cargile membrane,[8, 20] derived from the mesentery of a sheep, is probably the best tissue for a prostheses. It is obtainable in drug stores as the fish-skin condom. It is non-irritating, moisture-proof, somewhat transparent, and has an inherent adhesive quality. It is applied to the perforation in adequate size (one-fourth to one-half larger than the perforation) one or two days after the epithelium along the edge of the perforation has been scarified or cauterized with trichloracetic acid. It is introduced on and maneuvered by a wet, cotton-tipped applicator and readily attaches itself to the drum. It may be applied with its opposing surface dry or to the perforation rimmed with an ointment such as scarlet red and may be allowed to remain indefinitely.

It is generally agreed that a prosthesis should not be used if the middle ear remains moist.

During the war, at Borden General Hospital, Chickasha, Oklahoma, an excellent opportunity to observe and treat perforations which were from one to six or more months old was available to Work[22] and his associates. In the clean, dry, non-marginal perforations, they learned to screen out those which would likely respond to definitive treatment from those which appeared doomed to failure. In the latter group were included those perforations which had a third dimensional factor. This third dimensional factor, according to Work, means simply that the periphery of the perforation lies in different planes. For example, the superior margin of a perforation can be pulled in by the inward displacement of the malleus' handle or the inferior margin can be displaced medially or laterally to such an extent that it becomes impossible for this part of the drum to unite with the upper portion.

Among those cases designated as suitable for treatment, they obtained a number of successful closures of large as well as small perforations with the use of Cargile membrane. They considered it more expedient to scarify the perforation edges with a knife rather than use chemical cautery before applying the membrane. The membrane was placed in position immediately after the scarification if the bleeding was not excessive.

That large perforations can heal without a discernible scar is worthy of emphasis. The appearance of both drums after healing in Case 1 is a good illustration of this. Alexander,[2] in discussing perforations without infection, stated," After the regeneration of the

drumhead is complete, it is quite impossible to recognize any trace of the site of the previous perforation." This statement, in our experience, should be amended to include some perforations with infection. It could be further amended to include certain nontraumatic perforations, according to Stinson,[20] who states that scars from an otitis media may disappear through the "escalatory action" of the drum epithelium.

The effects of blast upon the auditory apparatus other than the ear drum have not been discussed because they have been reported elsewhere.[5, 7, 10, 14, 15, 19] Reports, however, on the reparative power of the ear drum in blast injuries have been meager. For this reason and because the knowledge gained from the experience with perforations sustained in wartime can be applied to the management of those of peacetime, this report on the repair of the ear drum in blast injuries has been presented.

SUMMARY

Complete healing of the tympanic membrane following blast injury occurred in many cases after almost total destruction of the pars tensa, provided that no part of the perforation reached the sulcus tympanicus.

Failure of the perforation to close was due, in some instances, to a third dimensional factor.[22]

Associated infection of the middle ear did not prevent closure of a large blast perforation.

The tympanic membrane, in some cases, healed without visible evidence of previous injury.

The use of prostheses increased the incidence of healed perforations.

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REFERENCES

1. Alexander, A. B.: Blast Injuries to the Ear, Med. Press & Circular, Vol. 209 (April 21) 1943.

2. Alexander, A. B.: Rupture of the Drumhead as a Wartime Injury, Brit. M. J. 2:195 (Aug. 9) 1941.

3. Bettington, R. H. B.: Injuries to the Ear Due to Blast, M. J. Australia 2:210-211 (Aug. 18) 1945.

4. Cantor, J. J.: Rupture of Tympanic Membrane Due to Blast; Mechanical Hypothesis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 54:554-567 (Sept.) 1945.

5. Colledge, L., and others (including Collier, J.): Discussion on Injuries of the Ear, Proc. Roy. Soc. Med. (Sect. Otol.) vol. 34, 1940-41.

6. Collins: Quoted by Henry.[11]

7. Craig, D. H.: Blast Injuries of the Ear, J. Laryng. & Otol. 59:443-456 (Dec.) 1944.

8. Fox, S. L.: On Closing Tympanic Perforations, South M. J. 38: 492-493 (July) 1945.

9. Greaves, F. C., Draeger, R. H., Brines, O. A., Shaver, J. S., and Corey, E. L.: An Experimental Study of Underwater Concussion, U. S. Nav. M. Bull. 41:339-352 (March) 1943.

10. Guild, S. R.: War Deafness and Its Prevention, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:70-76 (March) 1941.

11. Henry, G. A.: Blast Injuries of the Ear, Laryngoscope 55:663-672 (Nov.) 1945.

12. Hoople, G. D.: An Otolaryngologist's Experience in World War II, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 55:121 (March) 1946.

13. Ireland, P. E.: Traumatic Perforations of Tympanic Membrane Due to Blast Injury, Canad. M. A. J. 54:256-258 (March) 1946.

14. Korkis, F. B.: Rupture of Tympanic Membrane of Blast Origin, J. Laryng. & Otol. 61:367-390 (July) 1946.

15. Machle, W.: The Effect of Gun Blast Upon Hearing, Trans, Am. Acad. Ophth. & Otol. (Nov.-Dec.) 1944.

16. McNaught, R. C.: Personal communication.

17. Palma, J., and Uldall, J. J.: Immersion Blast Injuries, U. S. Nav. M. Bull. 41:3-8 (Jan.) 1943.

18. Palmer, C. W.: Blast Perforation of Ear Drums, Brit. M. J. 2:233 (Aug. 21) 1943.

19. Silcox, L. E., and Schenck, H. P.: Blast Injury of the Ears, Arch. Otolaryng. 39:413-420 (May) 1944.

20. Stinson, W. D.: Closure of Perforations of Membrana Tympani with Cargile's Membrane, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:178-181 (Mar.) 1941.

21. Stinson, W. D.: Reparative Processes in the Membrana Tympani, Arch. Otolaryng. 24:600 (Nov.) 1936.

22. Work, Walter: Personal communication.

23. Wittmaack, K.: Ueber die Normale und Pathologische Pneumatization des Schlafenbeines, Jena, G. Fischer, 1918.

24. Dunlap, A. M, and Schuknecht, H. F.: Closure of Perforations of the Tympanic Membrane, Trans. Amer. Laryng., Rhin. & Otol. Soc. 1947, pp. 215-227.

XI

BRONCHOGRAPHY IN BRONCHIECTASIS IN CHILDREN

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Bronchiectasis is one of the most common diseases of the chest, even more prevalent than tuberculosis. It is probable that in most cases the initial injury or insult to the lung occurs during childhood. Pediatricians who have made a study of this disease are impressed by the frequency with which the onset follows a protracted convalescent period from one of the common childhood diseases, particularly measles and pertussis or streptococcal or influenzal pneumonia.

Brenneman[1] points out that in childhood bronchiectasis is an evolution from a prebronchiectatic state, recognizable by the triangular shadow at the base of the lung in the x-ray film. Such a shadow, usually associated with a shift of the mediastinum, has been shown to be due to atelectasis. Obstruction to the lumen of a bronchus produces atelectasis, and bronchiectasis then develops in the atelectatic area. Obstruction of a bronchus is probably most commonly caused by secretions. Other factors are foreign bodies, benign tumors, granulations and stenoses from various causes.

The role of sinusitis as an etiological factor in bronchiectasis is still a controversial question. Riggins[2] states that a review of 100 cases of bronchiectasis seen at Lenox Hill Hospital showed that definite chronic sinus disease was present in 30 per cent. An analysis of the histories of these 30 patients showed that two-thirds had symptoms or findings, or both, of bronchiectasis from several months to several years before they developed chronic sinus disease. Diamond and Van Loon[3] found that the incidence of sinus disease in bronchiectatic patients varied with the age of the patient. "Co-existing sinusitis was noted in only 44 per cent of the children seen before the age of five years, whereas it was found in 66 per cent of those whose condition was diagnosed at a more advanced age." Walsh and Myers[4] found sinus disease in association with bronchiectasis in 66.8 per cent of a series of 217 cases. Certainly the two conditions, bronchiectasis and chronic sinusitis, are so frequently associated that one

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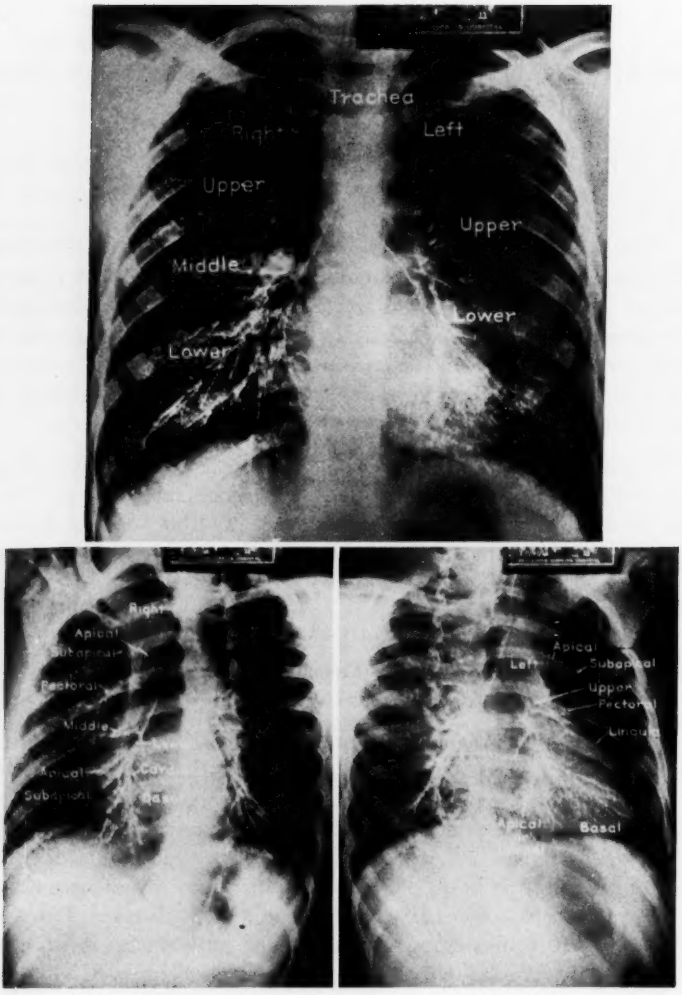

Fig. 1.—Normal bronchogram in a child eleven years old. (Courtesy of Dr. Janet Hardy.)

is forced to the conclusion that in some way there does exist a cause-and-effect relationship between the two.

Once the disease is established it is irreversible. If not too widespread, it is a surgically curable disease. There apparently is no relation between the severity of symptoms and the number of lobes or lungs involved. Before treatment is instituted, not only must the existence of the disease process be established but the extent and the location of the process must be accurately determined. In a large number of patients whose history and physical findings would lead one to the presumptive clinical diagnosis of bronchiectasis, actual dilatation of the bronchi cannot be demonstrated.

For confirmation of the diagnosis and accurate localization of the disease process one must rely on the bronchogram. All other examinations are adjuncts to bronchography or are done in the preparation of the patient for this procedure. Bronchoscopy, though essential and though it may yield information strongly presumptive of a diagnosis of bronchiectasis, is rarely confirmatory. It is rare indeed that one can see dilatation of a bronchus through the bronchoscope.

Sicard and Forestier were the first to use iodized oil in diagnostic bronchography. Since that time there have evolved several methods for introduction of the oil into the bronchi. It is unfortunate, however, that many hospitals rarely attempt bronchography on the small child, feeling apparently that the difficulties are too great.

In some clinics, with the patient under local anesthesia, the tongue is held well forward and the oil is simply dripped over the base of the tongue filling the valleculae which overflow, allowing the oil to drain down into the trachea and bronchi. The patient must refrain from swallowing during the procedure. This method obviously cannot be successfully used when working with small children.

Wishart[5, 6] in two very excellent articles has presented his technique of bronchography, whereby the iodized oil is injected through a bronchoscope with the child under general anesthesia. In his hands this technique has proven so satisfactory that it is still in use with slight modifications at the Hospital For Children, Toronto, Canada.

Jackson and Bonnier[7] described the catheter technique in 1937 which is now probably most generally used for adults in the majority of clinics throughout the country. The authors recommended that the method could, with slight modification, be made adaptable to children.

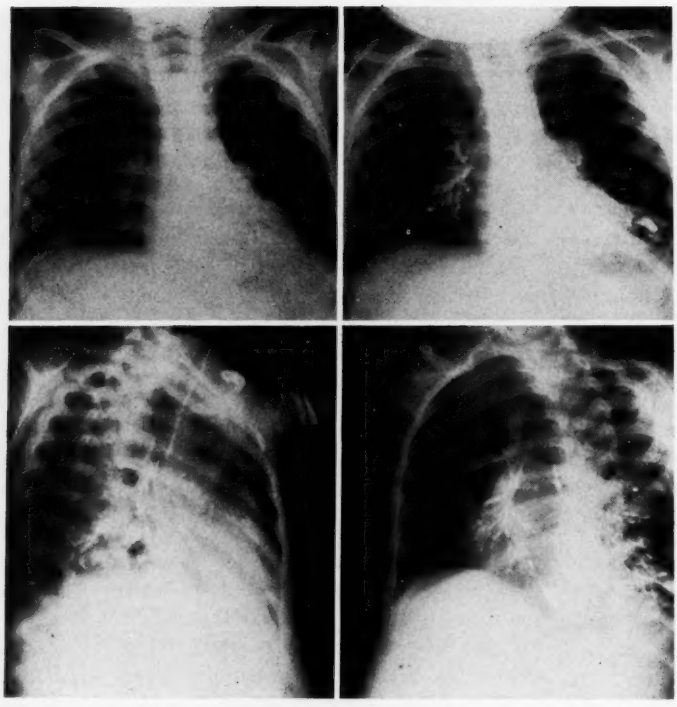

Fig. 2.—Bronchiectasis in the left lower lobe in a child two and one-half years old.

At The Johns Hopkins Hospital, the diagnosis of bronchiectasis in children and the preparation of these children for operation have been combined responsibilities of the pediatrician, the otolaryngologist and the radiologist. The primary responsibility rests with the pediatrician who conducts the child through the various diagnostic procedures and correlates the results. The preliminary work-up includes a thorough history, physical examination, ear, nose and throat consultation and routine x-ray examination of the chest. A bronchoscopy is then done. No matter how sure the presumptive diagnosis of bronchiectasis one should never lose sight of the underlying etiological factor which produces the condition, namely, obstruction of the bronchus. Only with the bronchoscope can the presence of an obstructive lesion be determined.

The preliminary work-up having been accomplished, the child is now scheduled for bronchography. If a satisfactory bronchogram is to be obtained, the bronchiectatic patient must be properly prepared by every means at our disposal in order to lessen the amount of secretion in the bronchial tree. Bronchi that are already filled with secretions cannot be filled with iodized oil. The child is placed on postural drainage. Penicillin is given by injection and in the form of an aerosol. Penicillin aerosol has been found to aid materially in temporarily decreasing the amount of secretions. Bronchoscopic aspiration, and repeated aspirations if necessary, are used to cleanse the tracheobronchial tree before the opaque medium is instilled.

In this hospital, the catheter technique and local anesthesia are used for children as well as adults. It is believed that bronchography under general anesthesia is an unnecessarily tedious and time-consuming procedure. Furthermore, there are certain inherent dangers to the patient and to the examining personnel in this method which cannot be avoided. If proper filling of the bronchi is to be obtained the cough reflex must be abolished. Under general anesthesia, this means that the level of anesthesia must be quite deep and maintained so while the bronchi are filled and pictures taken and this has to be accomplished with the child in a completely darkened room. This should give pause to even the best trained anesthetist. The use of ether anesthesia in a small room where x-ray equipment is being used is not calculated to add to the operator's peace of mind.

At this institution, bronchograms are customarily made in the afternoon. The child receives nothing by mouth during the preceding six hours. Approximately one and one-half hours prior to the operation, one of the barbiturates is given. Adequate dosage of the barbiturate is considered doubly important because of its seda-

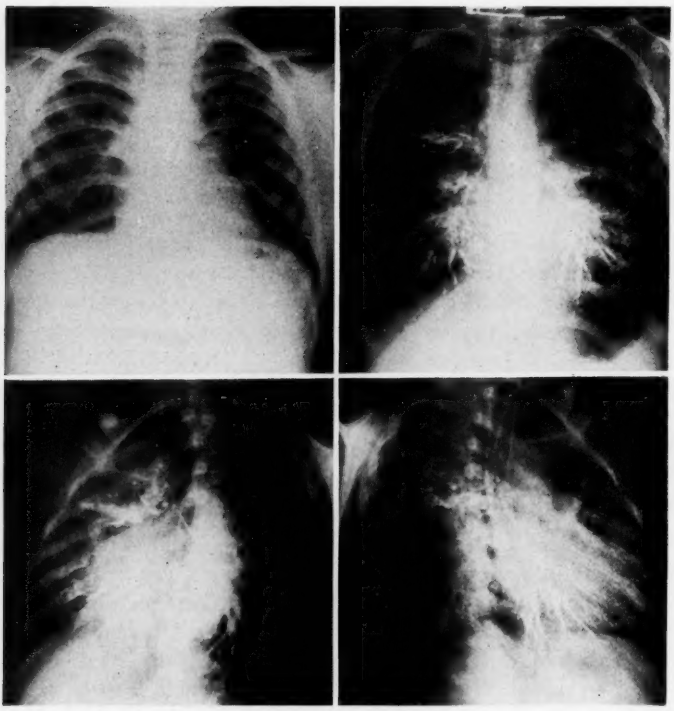

Fig. 3.—Saccular bronchiectasis localized in the right upper lobe in a girl six years old.

tive effect and because it may help to offset any possible toxic reaction to the pontocaine which is used as a local anesthetic. On call to the bronchography room, the patient receives atrophine and morphine sulphate. All or part of the preoperative medication is omitted on certain occasions depending on the age and the condition of the patient.

The child is seated on a chair facing the anesthetist; a stool is placed on the chair for small children in order to raise them to the desired height. The pharynx is first sprayed lightly two or three times with 0.5% pontocaine. Between sprays while waiting for the anesthesia to take effect, one side of the nose is anesthetized by passing a cotton-tipped applicator moistened with 2% pontocaine along the floor of the nose. This must be done two or three times before sufficient anesthesia is obtained to permit passage of the nasal catheter. As soon as the pharynx begins to feel numb, a cotton-tipped curved metal applicator moistened with 2% pontocaine is used. With the child or an assistant holding the tongue forward, the applicator is passed over the posterior pharyngeal wall and into both pyriform sinuses; the purpose of the latter being to reach the superior laryngeal nerves which are the chief sensory nerves supplying the larynx. The blockage of this pathway abolishes the cough reflex which Jackson has so aptly termed the "watchdog of the lungs." Working slowly and while using the laryngeal mirror, one can anesthetize the vocal cords and eventually pass the applicator between the vocal cords without inciting the cough reflex. From 0.5 cc. to 1 cc. of 0.5% pontocaine is now instilled into the trachea. The solution is drawn into a small syringe to which is attached a curved metal catheter, and while the glottis is visualized in the laryngeal mirror, the pontocaine is dropped, drop by drop, between the vocal cords.

The nasal catheter is now passed. A flexible rubber nasal catheter, to which is attached a metal adapter to fit a standard 20 cc. syringe, is used. A catheter must be selected that is small enough to pass readily between the vocal cords and yet leave an adequate airway, and at the same time, large enough to facilitate directing it into the trachea and assure that it will remain in place even though the patient should cough. The catheter is passed through the side of the nose which has been anesthetized and frequently may be passed into the trachea blindly, without the use of the laryngeal mirror, simply by holding the child's tongue forward, and having him breathe deeply and slowly while advancing the catheter. This should be done on inspiration only. The patient should be cautioned to avoid talking or swallowing. If the catheter does not readily enter the trachea, the

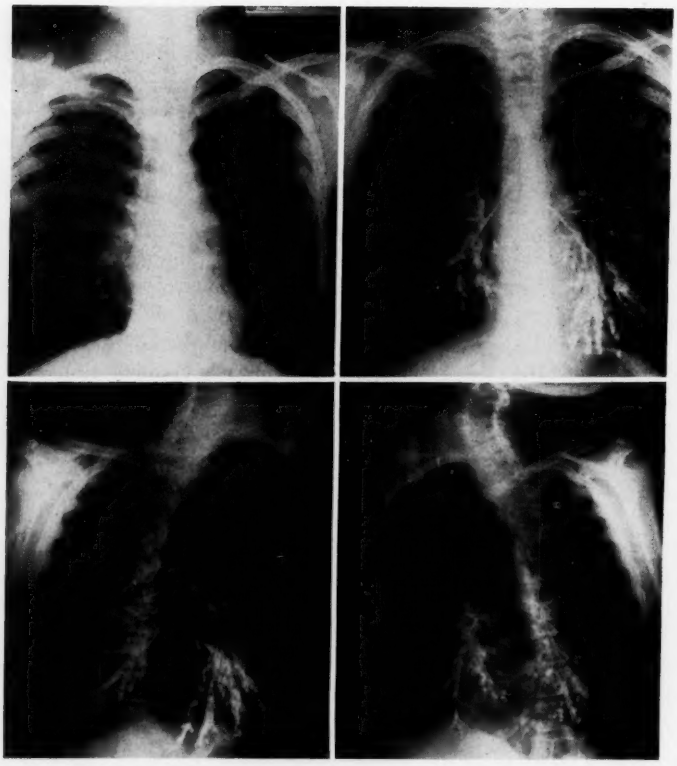

Fig. 4.—Extensive bronchiectasis involving both lower lobes and the lingula. The patient is twelve years old.

tube should be grasped through the open mouth with dressing forceps and, while visualizing the glottic chink in the mirror, directed between the cords into the trachea. With some practice, one soon becomes quite adept at this and little difficulty is experienced.

It is rare indeed that a child four years of age or over cannot, with the exercise of some patience, be intubated by the above procedure. Occasionally with smaller children and more rarely with the older ones, more drastic procedures become necessary. In that case, the child is restrained and the catheter is passed through the nose into the oropharynx. The larynx is then exposed with a laryngoscope, the tip of the catheter is grasped with forceps and directed into the larynx.

Immediately after the intratracheal tube is in place and if an anesthetic was not previously instilled, from 0.5 cc. to 1 cc. of 0.5% pontocaine is injected through the tube into the trachea. By closing the tube with the finger one can prevent the solution's being expelled by the cough which usually follows. If the 0.5% pontocaine solution was previously instilled, usually no more is required at this time; however, just before the child is placed on the fluoroscopic table, a few drops of the pontocaine are again injected into the trachea through the catheter. If no cough is produced, no further instillation is necessary.

There is no set rule as to the total amount of pontocaine to be used. The amount required is the amount necessary to abolish the cough reflex. This may be as little as 1 cc. to 2 cc. of the 0.5% solution in some children; others will require as much as 4 cc. If an excessive amount of secretions remain in the tracheobronchial tree, it will be difficult or impossible to secure an adequate anesthesia. We make it a general rule never to instill more than 4 cc. of the 0.5% solution.

The child is now placed on the tilt-table under the fluoroscope and the level of the catheter is first checked. The tip of the catheter should not be pushed down into either main bronchus. Satisfactory filling may be obtained with the tip of the catheter just above the level of the carina. As the injection of the oil is begun, the child's feet must be slightly lower than the head to prevent the oil flowing back into the trachea. By rolling the patient to one side or the other, the opaque medium may be made to flow into either main bronchus and, by raising or lowering the foot of the table as required, the oil can be directed into any lobe. The progress of the filling is watched under the fluoroscope.

All lobes of both lungs are mapped at one sitting. This obviates the necessity of having the child return for additional study unless for some reason adequate filling is not obtained. The bronchogram is not considered satisfactory unless all lobes are mapped. On an average, 15 cc. of iodized oil are used for each patient.

The filling completed, the catheter is quickly withdrawn and the exposures are made immediately. The anteroposterior and both right and left anterior oblique views are taken routinely. Adequate segmental localization of the disease process can be made from these three exposures.

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REFERENCES

1. Brenneman, Joseph: Bronchiectasis as the Pediatrician Sees It, Dis. Chest 9:39-49 (Jan.-Feb.) 1943.

2. Riggins, H. McL.: Present Concepts of Pathogenesis, Morbidity, Mortality and Treatment of Bronchiectasis, Dis. Chest 9:5-23 (Jan.-Feb.) 1943.

3. Diamond, Sidney, and VanLoon, E. L.: Bronchiectasis in Children, J. A. M. A. 118:771-778 (March 7) 1942.

4. Walsh, J. W., and Myers, O. O.: Co-existence of Bronchiectasis and Sinusitis, Arch. Int. Med. 61:890 (June) 1938.

5. Wishart, D. E. S.: Bronchoscopy in the Diagnosis and Treatment of Bronchiectasis in Children, J. A. M. A. 120:1181-1186 (Dec. 12) 1942.

6. Wishart, D. E. S.: Bronchography in Bronchiectasis in Children, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 56:404-415 (June) 1947.

7. Jackson, C. L., and Bonnier, M.: Technique of Bronchography, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 46:771-785 (Sept.) 1937.

XII

LARYNGEAL EDEMA IN EPIDEMIC PAROTITIS

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Edema of the larynx occurring in epidemic parotitis, although rare, is potentially a very dangerous complication. Descriptions of two cases of laryngeal edema occurring in mumps which were observed in the out-patient clinic at The Johns Hopkins Hospital follow:

CASE 1.—M. A., a colored female, aged 32, was examined April 23, 1947. Her chief complaints were swelling in the left side of her neck and pain on swallowing. These symptoms began three days previously when she noticed pain and swelling of the soft tissues of the neck just below the left mastoid process. During the succeeding two days she experienced chilly sensations, felt feverish, and swellings in the parotid and submaxillary areas developed. The day before admission the patient noticed pain on swallowing and became slightly hoarse. Her hoarseness had disappeared on the day of examination, but she stated that it made her voice tired if she talked very much. It was learned that the patient's sister had visited her on April 6, 1947, and that at that time the sister had had a painless swelling of the cheek.

Physical examination at the time of admission revealed a temperature of 100° F. There was moderate swelling of the left zygomatic, parotid and submaxillary areas and the skin overlying these areas was slightly red, felt warm and was slightly tender on pressure. A definite fullness of the peritonsillar tissue on the left was noted, but no infection of the tonsils was seen. The laryngeal airway was free and the vocal cords appeared normal. There was marked edema of the mucous membrane overlying the left arytenoid process, extending out into the aryepiglottic fold. The blood count was as follows: red blood cells 4,600,000, hemoglobin 13.5 grams, white

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blood cells 3,400, young forms 14 per cent, polynuclears 58 per cent, basophiles 2 per cent, lymphocytes 24 per cent, monocytes 2 per cent. The sedimentation rate was 23.

On the day following admission, both submaxillary glands were swollen. Mirror examination of the larynx showed some reduction in the edema on April 25 The laryngeal edema had entirely disappeared on April 29, 1947. The patient did not receive any sulfonamide or penicillin therapy.

CASE 2.—R. B., a colored male, aged 41, was examined in the out-patient clinic on July 24, 1947. The patient's chief complaint was a painless swelling of the right side of his neck below the right mandible of three days' duration. The patient denied fever or chills and with the exception of the presenting complaint had been well. He denied having had mumps, but stated that two of his children were recovering from mumps at that time.

The temperature was normal. The right submaxillary gland was moderately swollen and slightly tender to touch; there was no discoloration of the overlying skin. Mirror examination of the larynx revealed definite edema of the mucosa of the right pyriform sinus and the interarytenoid area of the larynx. There was marked edema of the soft tissues overlying the sternum which produced a fullness of the suprasternal notch. The blood count was as follows: white blood cells 7,900, young forms 1 per cent, polynuclears 39 per cent, lymphocytes 41 per cent, monocytes 14 per cent, eosinophiles 1 per cent, atypical forms 3 per cent.

The patient was advised of the necessity of bed rest in order to prevent complications and was sent home. During the following week he was visited daily by a member of the visiting nurses service. There was no increase in the severity of his symptoms, and he remained free of fever. No hoarseness or dysphagia was present at any time. A week later when the patient returned to the out-patient clinic, there was no edema of the larynx and only a slight bilateral enlargement of the submaxillary glands. At no time did the patient receive any sulfonamide or penicillin therapy.

DISCUSSION

The diagnosis of an isolated case of epidemic parotitis, particularly in the adult, is not always easy. A careful history is important; this was especially borne out in the two cases reported. In the first, the history of contact was elicited only after the tentative diagnosis of mumps with laryngeal edema had been made. In the second in-

stance, the edema of the larynx was noted during the routine examination of the nose and throat. This, coupled with the swelling of the submaxillary gland and the presternal edema, immediately brought to mind the possibility of mumps.

Mumps presents a great variety of complications. The more common ones include: orchitis, phlebitis, meningitis, meningo-encephalitis, pancreatitis, deafness, nephritis and conjunctivitis. Much rarer complications are optic neuritis, choroiditis, iritis, endocarditis, pericarditis, presternal edema and laryngeal edema. Apparently presternal and laryngeal edema are quite uncommon. It is probable, however, that both conditions occur more frequently than a review of the literature would lead one to believe and often are simply overlooked. The etiology of both complications is obscure, but it is probable that each is the result of some obstruction to the normal lymphatic drainage. This may be secondary to the pressure of the swelling.

Although edema of the larynx is mentioned as a complication of mumps by some textbooks,[1] a search of the literature revealed no cases reported in America. Reverchon, Worms, and Delater[2] described six cases in France in 1922. Of these six cases, three were fatal, a tracheotomy was required in one, and two were mild. Reverchon,[3] in 1934, again reported a case in which a tracheotomy was required, and in 1935[4] added four more cases of edema of the larynx during epidemic parotitis.

Of interest is a report by MacLeod[5] on a survey of 694 cases of mumps during 1919. The study was made on members of the Canadian forces overseas in England. He listed laryngitis and aphonia as complications in eight patients. Accepting these symptoms as diagnostic of laryngeal involvement, the incidence in this particular series was about 1.15 per cent. Unfortunately no examination of the larynx was made.

Radin[6], in 1918, reported presternal edema in mumps. Barker[7] reported three cases of this complication from observation of 438 cases in 1943. He also noted that one of these patients complained of hoarseness and difficulty in swallowing. The consultant otolaryngologist stated, "There is edema of the epiglottis and the right lateral wall of the hypopharynx, but very little of the larynx; though the latter could not be completely seen." The largest series of cases with presternal edema was reported by Gellis and Peters[8] in 1944. In 502 cases of mumps, they observed 30 cases of presternal edema, an incidence of 6 per cent. Of the 30, two had short periods

of dysphagia and one complained of hoarseness. In contrast to the report by MacLeod, this would indicate an incidence of laryngeal edema in only .2 of 1 per cent. Dysphagia may well be secondary to laryngeal edema, and if we accept this assumption, then the incidence would be about .5 of 1 per cent in this series.

The complete lack of reported cases of laryngeal edema as a complication of epidemic parotitis, in this country, leads one to believe that the recognition of this important complication is usually missed. Mumps should be considered as a possible diagnosis in all cases of unexplained laryngeal edema, particularly when associated with salivary gland enlargement.

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REFERENCES

1. Rolleston, J. D., and Ronaldson, G. W.: Acute Infectious Diseases, C. V. Mosby Co., 1940, p. 250.

2. Reverchon, L., Worms, G., and Delater, L.: Contribution á l'anatomie pathologique des oreillons. Mort par oedeme du larynx au cours de cette infection, Paris Med. 43:471-474, 1922.

3. Reverchon, L.: Edema, Submaxillary Mumps with Perilaryngeal Edema in Adult; Use of Emergency Tracheotomy in Case, Lyon Med. 154:702-706 (Dec.) 1934.

4. Reverchon, L.: Laryngeal Edema During Parotitis, Ann. d'Otolaryng. 1087-1096 (Oct.) 1935.

5. MacLeod, G.: Mumps in Adults, Brit. M. J. 2:742-743 (Dec.) 1919.

6. Radin, M. J.: The Epidemic of Mumps at Camp Wheeler, Arch. Int. Med. 22:354-369 (Sept.) 1918.

7. Barker, C. S.: A Complication of Mumps—Swelling Over the Manubrium Sterni, Can. M. A. J. 48:22-23 (Jan.) 1943.

8. Gellis, S. S., and Peters, M.: Mumps With Pre-Sternal Edema, Bull. Johns Hopkins Hosp. 75:241-250 (Oct.) 1944.

XIII

PENICILLIN TREATMENT OF NERVE DEAFNESS DUE TO SYPHILIS

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In order to evaluate properly a method of therapy in syphilitic deafness, repeated objective observations over a prolonged period of time are necessary. No such study has appeared in regard to penicillin treatment. It is the purpose of this publication to present detailed studies of the hearing in patients with syphilitic deafness treated with commercial penicillin mixtures since September 1943 by the Johns Hopkins Hospital group. (Several similar cases treated with penicillin plus induced malarial therapy will be reported later.)

At the outset it was arranged that quantitative tests of the hearing should be carried out by the staff of the Otologic Research Laboratory. The syphilitic infection itself, as well as the general medical problems presented by these patients, was the responsibility of members of the Syphilis Clinic.

Patients with early syphilis were admitted to the study if they had lesions in which Treponema pallidum could be demonstrated by darkfield examination, a rapidly rising titered Eagle serum flocculation test for syphilis, or if the cerebrospinal fluid gave a strongly positive complement fixation reaction for syphilis, together with evidence of meningeal inflammation as shown by increased cell and protein determinations. Individuals with late syphilis (longer than two years' duration), whether congenital or acquired, were selected primarily on the basis of clinical findings. In the absence of ob-

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jective physical or psychiatric abnormalities, the requirement for inclusion in this study was that the serum flocculation test or the cerebrospinal fluid or both be positive. Patients were chosen in all instances with great emphasis on availability for follow-up examinations.

The diagnosis of syphilitic deafness was made when these patients showed a definite perception type of hearing impairment that could not be explained by the patient's age or other factors. Sudden onset and rapid progression of the deafness, together with marked shortening of the bone conduction time, strengthen the diagnosis. Unilateral deafness, as is well known and recently emphasized by Moore,[1] does not rule out syphilis as the cause. As a rule the onset of the deafness corresponded closely with the development of other syphilitic manifestations.

All individuals selected were hospitalized for the entire treatment period. Prior to the initiation of therapy complete case histories were obtained and physical examinations done. Blood and cerebrospinal fluid specimens were taken. The ears were carefully examined. The hearing was tested in a soundproof room with a Western Electric Company 1-A audiometer. Thresholds were determined for 14 frequencies, namely: 32, 64, 128, 256, 512, 1024, 2048, 2896, 4096, 5793, 8192, 10,321, 13,004, and 16,384 cycles per second. Spoken voice was tested with the Western Electric Company 4-A phonographic audiometer using the record with two-digit numbers. Weber and Rinne tests were performed with a 512 d.v. steel fork. Masking was used routinely for determination of bone conduction time, in the phonographic test and also, whenever it was indicated, in the audiometer test. All tests were done by one examiner. No attempt was made to evaluate vestibular function except when suggestive subjective complaints were noted.

Treatment consisted of intramuscular injections of commercial sodium penicillin in aqueous solution every three hours night and day until the arbitrarily selected total dosage had been administered. In general, smaller amounts were employed early in the study and increased as experience accumulated and a greater supply of the antibiotic agent became available. Prior to discharge from the ward, the general examination, the hearing tests and the laboratory tests for syphilis were repeated. The patients were then followed at frequent intervals in the Out-Patient Clinic in an attempt to evaluate the permanency of the results already obtained and to discover possible late changes.

A total of 13 patients with deafness due to syphilis have been thus followed. For purposes of comparison, eight patients who had neurosyphilis without hearing impairment and three who had neurosyphilis and loss of aural acuity due to middle ear disease were also studied.

Patients ranged in age from 11 to 55 years. The sexes were approximately equally represented and the colored race predominated, as is true of the general clinic census.

The patients with syphilitic deafness were divided into two groups according to the duration of the disease: Group I included all with early syphilis, Group II, those with late syphilis. Group III consisted of 11 patients employed as controls.

Group I. — Patients with Perception Deafness Complicating Acute Syphilitic Meningitis. The five patients in this category had one definite feature in common: the deafness was of recent origin. The duration of symptoms varied from eight days to a maximum of one month. Three of the individuals regained normal hearing in all respects, as proved by audiometric study, within 13, 85 and 177 days after the initiation of therapy and are reported in detail. Another patient showed significant improvement after 12 days; 1,114 days after the beginning of treatment the hearing in both ears was within the 20 decibel region and the loss for spoken voice was reduced from 25 and 20 per cent to 6 and 3 per cent. Perception by bone conduction that was originally missing returned and was only moderately shortened at the latest test. The last patient of this group showed definite improvement 63 days after treatment. Subsequent tests could not be obtained but the hearing probably returned to normal as based on the patient's statement 641 days after penicillin therapy when seen in the Out-Patient Clinic. Cases 1 and 3 showed clearly the sustained improvement in aural function following penicillin administration. In all three patients other cranial nerves were simultaneously involved. One year after the original illness one patient (Case 2) underwent an asymptomatic neurorecurrence without involvement of the auditory nerves.

Case 1.—(J. H. H. 342183) A 30-year-old Negro was first seen in the Syphilis Clinic after clinical and laboratory studies elsewhere had established the diagnosis of acute syphilitic meningitis as manifested by paralyses of the third, fourth, seventh and eighth cranial nerves on the right, and right-sided hemiparesis, all of four weeks' duration. A pretreatment audiogram showed that the hearing was normal on the left but that there was diminished acuity in

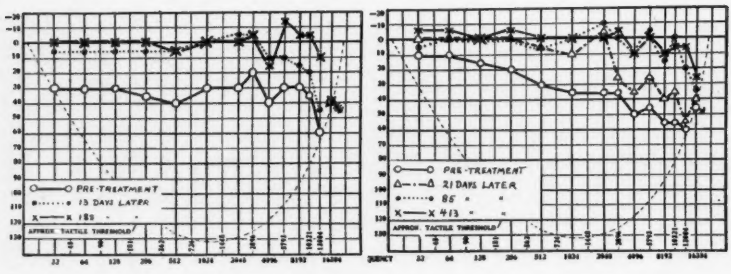

Fig. 1, Case 1.—Right Ear.

Fig. 2, Case 3.—Left Ear.

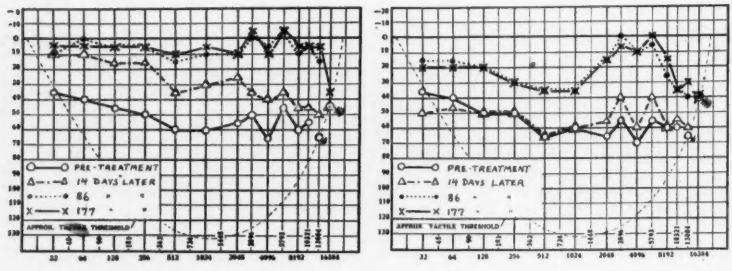

Fig. 3, Case 2.—Left Ear.

Fig. 4, Case 2.—Right Ear.

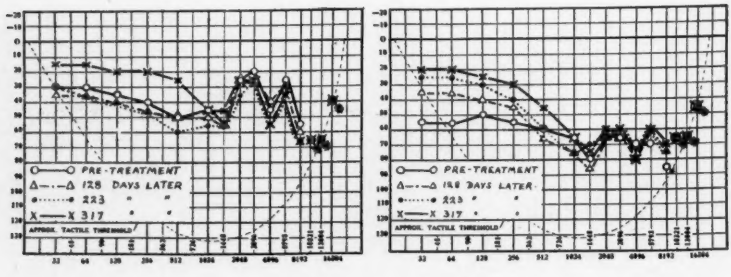

Fig. 5, Case 4.—Left Ear.

Fig. 6, Case 4.—Right Ear.

the right ear for all frequencies tested (Fig. 1). The Weber test was referred to the left from the glabella and the vertex but not from the chin. The Rinne test revealed that air conduction was greater than bone conduction in both ears. On the right, the fork was heard by air conduction for 49 seconds; on the left it was heard for 70 seconds. With masking of the opposite ear at 75 decibels, the fork was heard by bone conduction for 15 seconds in the right ear, 55 seconds in the left. The phonographic hearing test for spoken voice revealed about 6 per cent loss on the right and normal hearing on the left.

Treatment consisted of 50,000 Oxford units of penicillin every three hours for 80 doses, a total of 4 million units. There was a mild febrile Herxheimer reaction. Thirteen days after the start of treatment distinct audiometric improvement was demonstrable in all frequencies tested. The Weber test was not referred. In the right ear, the fork was now heard by air conduction for 96 seconds. With masking, the fork was heard by bone conduction for 27 seconds on the right, 40 seconds on the left. No loss for the spoken voice could be demonstrated in either ear by means of the phonographic audiometer.

When the patient was seen in the Clinic less than three months later, no clinical residuals of the illness could be found; the hemiparesis had disappeared. Serologic and cerebrospinal fluid negativity was achieved by 188 days after the start of therapy and an audiogram revealed essentially normal hearing. There has been no evidence of relapse during an observation period of 410 days.

CASE 2.—(J. H. H. 305141) An 18-year-old Negress was admitted to the ward because of headaches and dizziness for two weeks, bilaterally diminished hearing and right-sided facial paralysis for one week. Blood and cerebrospinal fluid examinations confirmed the diagnosis of acute syphilitic meningitis. Prior to treatment the audiogram showed marked diminution of hearing acuity in both ears for all frequencies tested (Figs. 3 and 4). The tuning fork, in the Weber test, was not heard from the midline points. In both ears the fork was heard by air conduction for 10 seconds but was not heard at all by bone conduction. The phonographic test revealed about 50 and 45 per cent hearing loss for the spoken voice in the right and the left ear, respectively.

Therapy consisted of 62 injections of 20,000 Oxford units each of penicillin, a total of 1.24 million units. Fourteen days later, after the completion of treatment, the audiogram showed great improve-

ment in acuity in the left ear. The Weber test was not heard from vertex or glabella but was referred to both ears from the chin. The fork was now heard by air conduction for 30 seconds in the right and for 45 seconds in the left ear. With masking of the opposite ear at 75 decibels, bone conduction times were 6 and 12 seconds in the right and the left ear, respectively. Hearing for spoken voice remained essentially unchanged in the right ear but improved to only 15 per cent loss on the left.

Re-examination at the end of 86 days showed the following changes: Weber test was referred from the midline points to the left ear. The 512 d.v. tuning fork was heard by air conduction for 30 seconds in the right and for 60 seconds in the left ear. With masking of the opposite ear, the fork was not heard by bone conduction in the right but was heard for 25 seconds in the left ear. Phonographic tests showed a loss in the right ear of about 12 per cent; hearing on the left was normal. Twenty-four days later the loss on the right had been further reduced to about 6 per cent.

The last examination, carried out 177 days after the start of treatment, showed minor changes only. Following treatment the facial weakness had disappeared entirely in less than one month. The serum flocculation test titer diminished and the cerebrospinal fluid became negative and has remained so during an observation period of 432 days.

CASE 3.—(J. H. H. 333674) A 29-year-old Negro was admitted to the ward for penicillin treatment of acute syphilitic meningitis as manifested clinically by paralyses of the left seventh and eighth cranial nerves which had been present for eight days. Hearing evaluation prior to treatment revealed diminished hearing, especially for the frequencies higher than 256 d.v. (Fig. 2). The Weber test was referred to the right. On the right, the 512 d.v. fork was heard by air conduction for 40 seconds and by bone conduction for 18 seconds. In the left ear the times were 25 and 7 seconds, respectively. The phonographic test for spoken voice showed no loss on the right, about 20 per cent loss on the left.

Treatment consisted of 50,000 Oxford units of penicillin for 80 doses, a total of 4 million units. There was no Herxheimer reaction. Twenty-two days after the start of these injections there was full restoration of hearing for the lower and middle frequencies. The other noteworthy changes included increases in perception time to both air and bone conduction in both ears and reduction of hearing loss for the spoken voice in the left ear to approximately 6 per cent (phonographic test).

The hearing was fully normal in an audiogram done 85 days after the initiation of therapy and remained normal in subsequent tests. In the last examination done 413 days after the beginning of treatment the Weber test was not referred. Air conduction time was now 91 seconds on the right and 92 seconds on the left. With masking, bone conduction times were 34 and 37 seconds for the right and left ears, respectively. No loss was demonstrable by means of the phonographic test.

This patient was treated for acute gonorrheal urethritis with oral penicillin (500,000 Oxford units) 336 days after the original therapy. Thirteen days later a routine cerebrospinal fluid examination was found to be strongly positive (neurorecurrence). Although the laboratory picture was that of distinct activity (370 cells per cu. mm. and 64 mg. of protein per 100 cc. of fluid), no clinical evidence of relapse was discernible nor was there diminution in hearing. He was retreated with penicillin with a total dosage of 8 million units.

Group II.—Patients with Nerve Deafness Associated with Late Syphilis. In this group deafness was, in general, of longer duration. Although the range extended from three weeks to six years, the mean was nearly two years. Three of the seven patients with acquired late syphilis (Cases 4, 5, 6) did attain increased hearing acuity which was remarkable in that the improvement became apparent long after the completion of treatment. Although none of the other four has improved, no further hearing loss developed in any of them during observation periods ranging from 81 to 514 days after the initation of therapy. The one patient in this study with late congenital syphilis (Case 7) has undergone progressive diminution of aural acuity since treatment.

CASE 4.—(J. H. H. 304152) A 44-year-old Negro was referred to the Syphilis Clinic because of a positive serum flocculation test and a history of a cerebrovascular accident. He had had a penile lesion 15 years before admission but had taken no treatment until two years previously, when he suffered a thrombosis of the left posterior inferior cerebellar artery. Although he had been immediately referred to this Clinic, almost a year elapsed before he was seen here. Noteworthy physical findings on admission to the ward were unequal, irregular pupils which reacted slightly, if at all, to light, and bilateral nerve deafness of five weeks' duration. The pretreatment audiogram disclosed diminished acuity for all frequencies tested (Figs. 5 and 6). The 512 d.v. steel fork was not heard from the midline points of the head but was perceived for

about two seconds from the mastoid processes. Air conduction was greater than bone conduction bilaterally both with and without masking of the opposite ear. Hearing for spoken voice (phonographic test) revealed about 55 per cent loss on the right, about 20 per cent loss on the left.

This patient was treated with 50,000 Oxford units of penicillin in each of 80 injections for a total of 4 million units. There was no Herheimer reaction. The next audiogram was made 128 days after the start of therapy (Figs. 5 and 6). At this examination the fork was not heard by bone conduction from the midline points or from either mastoid process; in the right ear it was heard by air conduction for 12 seconds and in the left ear for 22 seconds. The phonographic test revealed about 53 per cent loss for the spoken voice on the right, about 50 per cent on the left.

Re-examination 223 days after penicillin therapy disclosed no significant changes but after 317 days improvement was apparent. The fork was heard from the midline points but not referred to either ear. The Rinne test showed that on the right the fork was now heard by air conduction for 32, and on the left, for 60 seconds. Bone conduction time, with the opposite ear masked at 75 decibels, was 3 seconds on the right and 15 seconds on the left. It was concluded that definite improvement, both in air and in bone conduction, had occurred subsequent to penicillin treatment.

CASE 5.—(J. H. H. 322004) A 38-year-old Negress was admitted to the ward for penicillin treatment of late meningovascular neurosyphilis. She had not previously been treated. Examination disclosed paralyses of the fourth, sixth, seventh and eighth cranial nerves on the right, all of three weeks' duration. Pupillary changes were characteristic of late syphilis. The pretreatment audiogram (Fig. 7) showed diminished hearing acuity on the right. The Weber test was referred to the left. The 512 d.v. tuning fork was heard by air conduction for 8 seconds in the right ear (with masking) and for 65 seconds in the left ear. With masking of the opposite ear at 75 decibels the fork was heard for 35 seconds on the left but was not heard on the right. Using the phonographic audiometer, spoken voice was not heard on the right, whereas there was no loss on the left.

This patient was given 60 injections, each containing 20,000 Oxford units of penicillin, for a total of 1.2 million units. There was a febrile Herxheimer reaction. As judged by the audiometer tests, improvement in hearing could be seen as early as eight days after the start of therapy. Examinations were repeated at 20 and

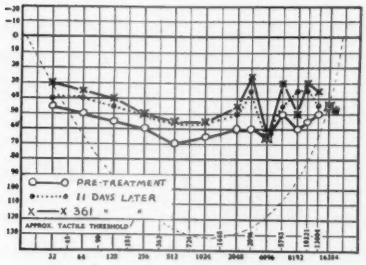

Fig. 7, Case 5.—Right Ear.

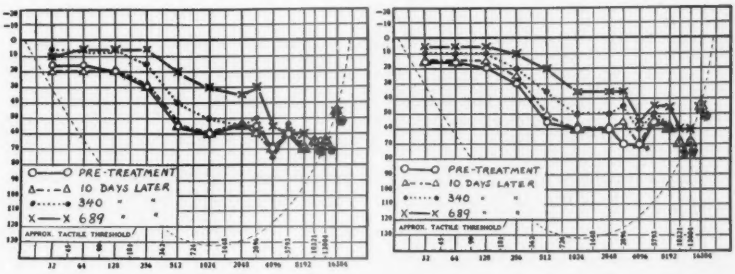

Fig. 8, Case 6.—Left Ear.

Fig. 9, Case 6.—Right Ear.

104 days. The latter test showed that the patient could hear the spoken voice (phonographic test) but not distinctly enough to understand it in the right ear. After 181 days this method disclosed only about 62 per cent hearing loss.

Re-examination at 504 days showed that the Weber test was no longer referred. The Rinne test revealed that the fork was heard by air conduction for 27 seconds in the right ear and for 85 seconds in the left ear. With masking of the opposite ear at 75 decibels, bone conduction time was nil on the right, 22 seconds on the left. The phonographic test showed about 55 per cent loss in the right ear and normal perception in the left.

The most recent aural examination, done 605 days after the initiation of penicillin therapy, showed minor changes except that the phonographic test revealed only 27 per cent loss on the right.

The last clinical examination disclosed only slight residual right-sided facial and right lateral rectus muscular weakness. Both the serum flocculation test and the cerebrospinal fluid test for syphilis were still positive but the latter was greatly improved. This case emphasizes the prolonged period of observation necessary for the evaluation of eighth cranial nerve lesions; improvement had taken place in the three-month interval between the last two examinations, roughly 17 and 20 months after therapy.

CASE 6.—(J. H. H. 141413) A 38-year-old Negro gave a history of inadequately treated syphilis of 25 years' duration and bilateral diminution of hearing for six months. His pretreatment audiogram showed bilateral impairment for all frequencies tested, but particularly for the higher tones (Figs. 8 and 9). The 512 d.v. steel fork was not heard from midline points or from either mastoid process; it was heard by air conduction for 16 seconds in the right and for 17 seconds in the left ear. The phonographic test revealed about 45 per cent loss for spoken voice on the right and about 40 per cent on the left.

Treatment consisted of 80 injections of 40,000 Oxford units each, a total of 3.2 million units. There was a febrile Herxheimer reaction. Re-examination at the end of treatment showed no significant change in the hearing but by 341 days after the start of therapy slight improvement in low and middle tone ranges was found. At that time the fork was not heard from midline points. The Rinne test revealed that the fork was heard by air conduction for 25 seconds in both ears. With masking of the opposite ear there was no bone conduction on either side. The phonographic test showed about 28 per cent loss on the right and about 40 per cent loss on the left. It was concluded that significant improvement had occurred.

The following case is presented to show the occurrence of progression of hearing loss in congenital syphilis despite penicillin therapy.

CASE 7.—(J. H. H. 127126) Nine months prior to penicillin therapy this 23-year-old Negress was referred to the Syphilis Clinic because of congenital syphilis complicated by inactive interstitial keratitis involving the left eye and bilaterally impaired hearing of about one year's duration. She was given alternating eight-week courses of weekly injections of mapharsen and bismuth subsalicylate. Unfortunately no pretreatment audiogram was obtained but hearing ability seemed to improve almost at once, from the subjective standpoint. During the eight months of this therapy, an exacer-

bation of the old interstitial keratitis occurred and she was admitted to the ward for penicillin treatment. It was found at this time that the Weber test was referred to the right and that air conduction was greater than bone conduction bilaterally.

She was given 62 injections of 20,000 Oxford units each, a total of 1.24 million units. No Herxheimer reaction was noted. The first thorough examination of aural function was done 52 days after the start of this therapy. Bilateral diminution of aural acuity was present for all frequencies tested. The tuning fork was not heard from the midline points. On both sides air conduction time was 15 seconds; the fork was not heard by bone conduction. Hearing for spoken voice (phonographic test) revealed about 47 per cent hearing loss on the right, about 41 per cent on the left.

A second evaluation carried out 731 days after the start of treatment revealed further loss of hearing, most marked in the higher frequencies. The other noteworthy changes were reduction of perception time for air conduction bilaterally to only 5 to 6 seconds and further hearing loss for spoken voice, as judged by the phonographic audiometer to about 59 per cent loss on the right and about 53 per cent on the left.

It is unfortunate that no quantitative pretreatment hearing tests were made, not only prior to penicillin but also prior to the arsenic-bismuth therapy first used. During antibiotic treatment the keratitis subsided entirely but the status of this patient's eighth nerve lesions has, as judged by these data, become significantly worse.

Group III.—Patients with Neurosyphilis, Treated with Penicillin, but without Syphilitic Deafness. In this control group were eight patients with no aural disease. Their examinations showed that penicillin did not render the auditory nerve susceptible to damage or itself reduce aural function in patients with syphilis. Two of these patients had acute syphilitic meningitis and five had late neurosyphilis. One of the former had an intense meningeal inflammation, with 1,450 cells per cu. mm. of fluid prior to treatment. The hearing in these patients was within normal range for their respective ages and no significant changes in aural acuity were found during observation.

The control group also contained three patients with impaired hearing apparently due to middle ear lesions. One had unilateral chronic otitis media with granulations; the other ear was normal. The second patient had bilateral secretory otitis media, and the third

had recurrent purulent otitis media. All three underwent improvement after their middle ear lesions cleared up under penicillin therapy.

DISCUSSION

At the beginning of this study no information as to the effect of penicillin in syphilitic deafness was available. Dosages were selected on an entirely experimental basis and altered as experience grew. Doses varied from 10,000 to 50,000 Oxford units. As prescribed, total dosages ranged from 600,000 to 4,000,000 Oxford units. Assays on various lots of commercial penicillin from different manufacturers at different times have shown that several species of penicillin in unknown proportions were employed in this series. It now seems likely that roughly one half the unitage administered was in the form of penicillin G or K.

Febrile Herxheimer reactions occurred in three of the patients with syphilitic deafness; a similar number of patients in the group with good hearing also exhibited this phenomenon. One patient in Group I had a marked subjective increase in hearing impairment together with mild vertigo during the first 24 hours of treatment. These symptoms spontaneously subsided and were attributed to an atypical Herxheimer reaction. No other unfavorable reactions to administration of the antibiotic were observed.

Based on experience with metal chemotherapy, alone or combined with fever, it was anticipated that syphilitic deafness associated with acute syphilitic meningitis should respond to the administration of penicillin if penicillin treatment was beneficial at all. With older methods it was often possible in such patients to prevent further hearing loss or even to restore fully aural acuity, if treatment was started soon after the onset of the impairment. Our studies show that penicillin treatment is fully as effective as the older methods in these cases.

In late neurosyphilis with deafness, chemotherapy has been admittedly of little or no help so far as hearing is concerned. Ciocco and Weinstein,[2] for example, state: "With the exception of deafness associated with early meningeal syphilis, it is concluded that antisyphilic treatment has no effect on hearing, either beneficial or detrimental." Penicillin-treated cases that belong in this category are reported in Group II. Three of these patients (Cases 4, 5, 6) improved under antibiotic therapy and maintained this improvement over the course of the observations. Four individuals appear to have received full protection from further damage to the aural mechanism; these may represent cases of spontaneous arrest. Only

one patient (Case 7), a congenital neurosyphilitic, developed an additional loss of hearing in the course of the study, not explained by increase in age or other factors and has to be termed a therapeutic failure. These results support the impression that penicillin therapy in deafness associated with neurosyphilis is of definite value and is superior to older methods of treatment.

No patient in the control series (Group III) with neurosyphilis and normal hearing developed deafness in the course of the observations; schedules of administration and total dosages of penicillin were comparable to those used in patients with deafness. Those with middle ear lesions obtained improved hearing with penicillin therapy, thus confirming the results of other workers who have dealt with middle ear disease in general.

The course of the hearing improvement varied according to the syphilitic lesion. The patients with acute syphilitic meningitis quickly noted definite improvement in the hearing. In the three patients with late neurosyphilis whose hearing improved, the change began after a much longer period of time and the hearing did not return entirely to normal.

The increase in acuity of the hearing started usually in the low tones and in some patients the maximum improvement occurred almost at once. This was followed later by gradual improvement in the perception of the higher frequencies. It is also of interest that a return of hearing by bone conduction occurred. Lund[3] reported no improvement of the bone conduction in a large number of patients treated with the older methods. Our material suggests that patients with acute syphilitic meningitis have a better chance than those with late neurosyphilis to recover hearing by bone conduction.

SUMMARY AND CONCLUSIONS

1. Results of experimental penicillin treatment of five patients with deafness associated with acute syphilitic meningitis and eight individuals with late neurosyphilis are reported. Commercial penicillin mixtures in aqueous solution were given intramuscularly in total dosages ranging from 600,000 to 4 million Oxford units. Eight neurosyphilitic patients with normal hearing and three with conduction deafness due to middle ear disease were studied under comparable conditions as a control series.

2. Hearing promptly became better in all patients with acute syphilitic meningitis, and the improvement was sustained. Of the seven patients with deafness associated with late neurosyphilis, four

had no change in hearing, either for better or for worse, and three had delayed but significant improvements in hearing acuity. The fact that hearing improved in approximately half of the patients of this group is particularly important, because with other methods of treatment it is rare to have hearing improve in patients with deafness associated with late neurosyphilis. One patient, a congenital neurosyphilitic, has undergone progressive hearing loss throughout the period of observation and has been the only treatment failure so far.

3. The increase in hearing acuity, when it occurred, started in the low tones. This was followed later by gradual improvement in the perception of higher frequencies. In several instances perception time for bone conduction, tested with a 512 d.v. fork, improved subsequent to penicillin treatment.

4. There is no evidence that the administration of penicillin has an unfavorable effect on hearing, either in patients with good hearing or in patients with hearing already impaired.

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REFERENCES

1. Moore, J. E.: Penicillin in Syphilis, Springfield, Ill., Charles C. Thomas, 1947, p. 265.

2. Ciocco, A., and Weinstein, A.: Involvement of the Eighth Nerve in Syphilis, Am. J. Med. Sci. 187:100, 1934.

3. Lund, R.: Neurolabyrinthitis syphilitica, Hab.-Schr. Kopenhagen, 1920.

XIV

RELATION OF DOSAGE TO STREPTOMYCIN TOXICITY

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In the short time since the introduction of streptomycin into clinical usage, a vast amount of information has been obtained on its therapeutic uses and its toxicity. Although much has been learned, there are still many gaps in our knowledge of how best to utilize this highly potent antimicrobic agent. It is well established that streptomycin is a valuable agent in the treatment of many acute and chronic infections for which no other potent drugs are available. The disadvantages of streptomycin are well recognized. These disadvantages are the frequency and severity of toxic reactions, and the rapid emergence of resistant strains of microorganisms. It is safe to state, however, that even if nothing further could be accomplished to minimize the inherent disadvantages of streptomycin, it would nevertheless remain an extremely valuable therapeutic agent.

There are several lines of attack which may be explored for the improvement in the clinical use of streptomycin. It is of interest to review some of the data which indicate the current trend in the attempts to prevent or to manage some of the important toxic reactions which follow the administration of streptomycin.

Following the discovery of streptomycin by Waksman and his associates,[1] the important preliminary studies in vitro and in experimental animals were carried out within a very short time. The clinical trials were made on the basis of these studies, and it has been gratifying to see how accurately the early laboratory studies delineated the field of usefulness of streptomycin. The dosage of streptomycin, the frequency of administration, and the duration of treatment in human infections had to be chosen arbitrarily, as is the case with all new drugs. After more than two years of experience, it is now apparent that the doses originally chosen were unnecessarily large. At the present time, in most clinics, considerably smaller

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doses are being employed. It is surprising, however, to consider how little is known about the therapeutic effectiveness of small doses or how much less than the usual doses one can administer in a given situation without altering the therapeutic results obtained. Two factors have contributed to the tendency to give large doses of streptomycin. First, the diseases for which it is effective are in general serious infections with a high rate of mortality for which no other effective antimicrobic agent is available. Confronted with these facts, clinicians are naturally inclined to neglect consideration even of relatively serious toxicity. Second, during streptomycin therapy there is a uniform tendency for the emergence of drug-resistant strains of bacteria. It has frequently been assumed that large doses of streptomycin given early in the course of therapy will influence this phenomenon. Despite the fact that there is little or no clinical evidence as yet to support this assumption, it continues to be a factor in causing clinicians to administer larger doses of streptomycin than may be required.

In addition to evaluation of the total dosage of streptomycin, the frequency of administration must be considered. Repeated intramuscular injections at intervals of three or four hours throughout the day and night have commonly been adopted in an effort to maintain concentrations in the body above a certain continuous minimum amount. This plan of administration is based on two assumptions: 1) that streptomycin is so rapidly absorbed and eliminated as to make frequent injections necessary in order to maintain "continuous levels"; and 2) that the "continuous levels" are necessary. There is clear evidence at hand that the first of these assumptions is incorrect, and suggestive evidence that the second assumption may also be unwarranted. Thus, it seems advisable to review some of the data on the toxicity of streptomycin in an attempt to determine what modifications of the present dosage schedules may be of value in reducing the frequency and severity of toxic manifestations.

First, it is of importance to know something of the absorption and distribution of streptomycin following single intramuscular injections in man. Within the last year, two accurate chemical methods of measurement of streptomycin in serum have been developed.[2, 3] In collaboration with Boxer and his associates of Merck and Co., we have studied the serum concentrations following intramuscular administration of doses ranging from 0.1 Gm. to 1.0 Gm. Details of these studies have been reported previously.[4] Several of the observations made are pertinent to this discussion. First, within the range of dosage customarily employed, it was found possible to predict

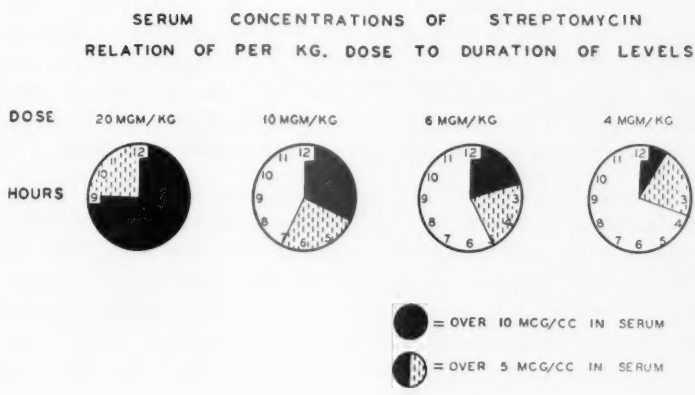

Fig. 1.

fairly accurately the amount of the serum concentration of streptomycin to be obtained, provided that the dosages were calculated in terms of the body weight of the patient. Moreover, within the dosage range of 4 to 20 mg. per kg. of body weight, it was observed that the rate of fall in serum concentration of streptomycin was proportional to the concentration. This rate of fall was actually not rapid, and on the average the time required for any given concentration to fall to half its original value was 2½ to 3 hours. The implications of these observations in regard to this discussion are: first, that the rational regulation of dosage schedules should be on the basis of the body weight of the patient; and second, that in all likelihood, long intervals between injections are permissible.

The persistence of streptomycin in the serum following a single injection is graphically illustrated in Fig. 1. The concentrations of 10 and 5 mcg. per cc. have been chosen arbitrarily for the purpose of illustration. It is known, however, that doses which seldom if ever achieve concentrations above 10 mcg. per cc. may be adequate in the therapy of many infections. In Fig. 1, assuming that a single injection has been given at 12 o'clock, the solid portion of each clock represents the average time the concentration is maintained above 10 mcg. per cc. after a single injection, and the solid plus the dotted portion represents the time the concentration is more than 5 mcg. per cc. The doses, from left to right, are 20, 10, 6 and 4 mg. per kg. In an average patient weighing 50 kg., these doses would be 1.0 Gm., 0.5 Gm., 0.3 Gm. and 0.2 Gm. One may conclude from these

figures that for the purpose of maintaining these relatively low serum concentrations, there is little to be gained from frequent small doses. The higher concentrations are, of course, not maintained to any significant degree with small doses. Although one can offer no conclusive proof, it would seem likely that the total daily dose of streptomycin may always be given in one, or at most two injections.

Among the numerous toxic reactions to streptomycin, at least two—impairment of hearing and impairment of vestibular function—appear to be related directly to the dose of the drug. In the studies of Hinshaw and of McDermott and their respective associates,[5, 6, 7] it has been observed that virtually all instances of deafness caused by streptomycin have been encountered in one of three situations: 1) in patients given excessively large doses—over 3.0 grams per day; 2) in patients with poor kidney function; and 3) in patients who receive intrathecal therapy for meningitis. Although the precise cause of deafness following treatment for meningitis is difficult to evaluate, in all of these situations the concentrations of streptomycin in the body and particularly in the central nervous system must necessarily have been unusually high. It now appears that it is seldom if ever desirable to administer these unusually large doses. Moreover, in patients with poor kidney function, particular care should be taken to avoid large doses. Obviously, other problems are encountered in regard to deafness, such as that of patients with partial hearing loss prior to the institution of therapy. Nevertheless, it would appear that in the vast majority of cases, satisfactory therapy with streptomycin may be given without undue risk of loss of hearing, with the possible exception of those patients who require intrathecal treatment.

The toxic effects of streptomycin on the vestibular apparatus have been a matter of great concern. The symptoms produced are distressing to the patient, although most individuals appear to make a satisfactory compensatory adjustment. The ultimate significance of this toxic manifestation is not fully known. In some patients marked improvement in vestibular function may occur, but the eventual outcome in most is uncertain. Whatever the eventual outcome proves to be, the occurrence of vestibular dysfunction during long-continued administration of streptomycin is sufficiently frequent and sufficiently severe to constitute one of the most troublesome toxic manifestations encountered.

It has become apparent relatively recently that the frequency of occurrence of vestibular dysfunction is related to the dosage of the drug and the duration of therapy. In Table 1 may be seen data in

TABLE 1.

RELATION OF STREPTOMYCIN DOSAGE TO IMPAIRMENT OF VESTIBULAR FUNCTION.

DAILY DOSE (GRAMS)	DAYS OF TREATMENT	PERCENTAGE OF PATIENTS WITH OBJECTIVE EVIDENCE OF IMPAIRED VESTIBULAR FUNCTION
3.0	42 to 120	90 - 100
2.0*	60	83
1.0 (5 doses)*	120	17
1.0 (2 doses)*	120	12
1.0 (1 dose)	42	15

*From data of Veterans' Administration.

two groups of patients treated at the New York Hospital,[8] and three groups of patients recently reported by the Veterans' Administration.[9] In the first column are shown the total daily doses, in the center column, the days of treatment, and in the third column, the incidence of impairment of vestibular dysfunction. It is recognized that data from the different sources may not be strictly comparable. They are meant only to illustrate that fewer instances of impairment of vestibular function occurred after the smaller doses, and to point out that the incidence of the reactions in our small group of cases was approximately the same as that in the larger and statistically more significant groups. In our own data are included only those patients whose responses to caloric stimulation, using the modified Kobrak test, were definitely normal prior to therapy and who had no response to maximal stimulation immediately after cessation of therapy. It should be emphasized that the particular test employed is of limited value in the quantitative measurement of gradations in impairment of vestibular function. The absence of response to caloric stimulation, however, would appear to be a definite objective measurement which may be used in the comparison of one group of patients with another.

As may be seen, in the first group of patients treated with 3.0 Gm. per day, virtually all developed vestibular dysfunction. In the second group of patients, those who received 2.0 Gm. per day, 83 per cent showed this reaction. The last three groups received 1.0 Gm. per day in one to five doses. The duration of treatment was from one and a half to four months. The incidence of vestibular

TABLE 2.

RELATION OF STREPTOMYCIN DOSAGE TO IMPAIRMENT OF VESTIBULAR FUNCTION.

All patients received single 1.0 gm. dose of streptomycin daily for 42 days.

DAILY DOSE MGM/KG.	NUMBER OF PATIENTS	NUMBER WITH NO RESPONSE TO CALORIC STIMULATION
13.4—19.0	14	0
19.0—25.6	13	4

dysfunction was approximately the same in these latter three groups, from 12 to 17 per cent, and in all it was significantly less than in the groups receiving the larger doses.

In view of the observation that the height of serum concentrations of streptomycin may be closely correlated with the dose per kilogram of body weight, it seemed of interest to calculate the dose per kilogram in this last group of patients. Each patient in this group received a single daily injection of 1.0 Gm. of streptomycin a day for 42 days. All patients have been excluded on whom caloric tests were not performed before and immediately after treatment, as well as for whom the dosage was variable, and who had obvious disease of the middle ear. There remains a total of 27 patients for whom the data are complete. When the dosage of streptomycin for the entire group was calculated in relation to the body weight, it was found that the dosage varied from approximately 13.0 to 25 mg. per kg. In Table 2, it may be seen that about half of the patients received 19 mg. per kg. per day or less, and the other half more than 19 mg. per kg. Four patients of the 27 were found to exhibit no response to caloric stimulation immediately after cessation of therapy, and two of the four complained of dizziness at some time during treatment. All four patients in whom definite vestibular dysfunction occurred were in the group which received the larger doses. It is obvious that the number of patients is small. Taken together with the data on the larger dose, however, it would appear to give an indication of the general level of dosage above which significant numbers of patients may be expected to develop vestibular dysfunction, and below which this toxic manifestation may be infrequent. As with other toxic reactions, it is likely that the occurrence of vestibular dysfunction may be modified by other factors, For example, it has been thought to occur more readily and to be in general more severe in elderly patients than in the younger age groups. Further information is necessary to clarify these points.

In addition to these and other toxic reactions to streptomycin which appear to be directly related to the dosage, a number of toxic reactions occur which appear to be manifestations of anaphylaxis, or hypersensitivity. These include drug fever, asthma, some of the skin eruptions, and the development of eosinophilia. The precise importance of these reactions in terms of interference with continuation of treatment is not known in all instances. Recently, some information has been obtained concerning a possible method of management of some of these reactions.

In the past year we have encountered four cases of drug fever, and one case of asthma caused by streptomycin; further therapy at the time was impossible because of the severity of the reaction. It is known that in some of these cases further therapy would probably have been possible two or three months later, but such an interval without treatment was undesirable. In all of these five patients it has been found possible to continue therapy without interruption with another drug, dihydrostreptomycin. Dihydrostreptomycin is closely related chemically to streptomycin, and, as far as tested to the present time, appears to have similar antimicrobic activity. Organisms sensitive to streptomycin are likewise sensitive to dihydrostreptomycin, and organisms resistant to one are also resistant to the other. Although only small amounts of this have been available for clinical testing, it appears to offer a promising possibility for use in some types of toxic reactions to streptomycin.

SUMMARY

The discussion has included consideration of some of the important toxic reactions to streptomycin and their relation to the dosage of the drug. From the data presented, it would appear feasible to effect a reduction in the frequency and severity of otic complications within the range of dosage now considered therapeutically effective, provided the dosage is calculated in terms of the body weight of the patient. Further information is highly desirable on the therapeutic effects of smaller doses than those commonly employed in an effort to determine how much can be accomplished in the further reduction of toxic manifestations. Mention was made of preliminary clinical experience in the use of another drug, dihydrostreptomycin, for the management of certain toxic reactions which are manifestations of anaphylaxis and not directly related to the dosage of the drug employed.

525 East 68th Street.

REFERENCES

1. Schatz, A., Bugie, E., and Waksman, S. A.: Production of Streptomycin by Actinomyces, Proc. Staff Meet., Mayo Clinic 19:537-548 (Nov. 15) 1944.

2. Boxer, G. E., and Jelinek, V. C.: J. Biol. Chem. (In Press).

3. Marshall, E. K., Jr., Blanchard, K. C., and Buhle, E. L.: Colormetric Methods for Determination of Streptomycin, J. Pharm. & Exp. Therap. 90:367-375 (Aug.) 1947.

4. Boxer, G. E., Jelinek, V. C., Tompsett, R., DuBois, R., and Edison, A. O.: Chemical Determination of Streptomycin in the Blood Following Intramuscular Injection, J. Pharm. & Exp. Therap. (In press).

5. Brown, H. A., and Hinshaw, H. C.: Toxic Reaction of Streptomycin on the Eighth Nerve Apparatus, Proc. Staff Meet., Mayo Clinic 21:347, 1946.

6. Farrington, R. F., Hull-Smith, H., Bunn, P. A., and McDermott, W.: Streptomycin Toxicity, J. A. M. A. 134:679-688 (June 21) 1947.

7. McDermott, W.: The Toxicity of Streptomycin, Am. J. Med. 2:5 (May) 1947.

8. Council on Pharmacy and Chemistry, Annual Report on Streptomycin, J. A. M. A. 135:641-642 (Nov. 8) 1947.

9. Veterans' Administration Meeting, St. Louis, Missouri, October 9-12, 1947.

XV

BENIGN LESIONS OF THE TRUE VOCAL CORDS

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So much attention has been given to malignancies lately that in discussing benign lesions of the vocal cords, one fears that he may detract from the great effort that has been made to educate the public, as well as the doctor, to the importance of early diagnosis in patients who have suspicious lesions. In no way do I wish to minimize or underestimate this importance. The fact is there are many more benign lesions of the vocal cords than malignant ones. The clinical picture of these lesions is not the final answer, however, and the entire tumor mass must be given the pathologist to aid us in our final diagnosis. Not only must we be able to assure the patient of the innocence of the lesion but we must be able to restore the voice to normal. In nearly every instance one should expect a normal voice after the removal of a benign tumor.

It is the purpose of this paper to discuss briefly the anatomy of the vocal cords, the various growths occurring on the vocal cords and then to discuss the treatment with regard to surgery, since in most instances removal is the only treatment.

Benign lesions of the vocal cords usually do not constitute a threat to life, but they do cause disability manifested by hoarseness and some benign lesions may be forerunners of malignant ones.

The more common lesions are papilloma, keratosis, vocal nodules, fibromata, contact ulcers, hematoma and leukoplakia. Some of the less common lesions are pachydermia, amyloid tumors, angioma, angiofibroma, cystoma, myoma, myxoma, thyroid tumors, teratoma and lipoma.

The true vocal cords are below the ventricular folds and extend from the angle between the lamina of the thyroid cartilage ventrally to the vocal process of the arytenoid cartilages dorsally. They meet anteriorly in what is known as the anterior commissure.

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The vocal fold is sharp and prominent and the mucous membrane which is stretched over it is very thin and firmly bound down to the subjacent ligament by elastic tissue. The color is pale, almost pearly white. In cross section the vocal fold is prismatic in form and its free border looks upward as well as medially. The muscles that go to make up the lateral portion of the vocal folds are the triangular, somewhat prismatic vocal muscles of each side which form with the thyro-arytenoid, a common muscular mass separated only with difficulty into its constituent portions.

The vocal muscle lies within the vocal fold, closely applied to the lateral aspect of the vocal ligaments, and receives its prismatic form from this adaptation. Of its three surfaces, the medial lies adjacent to the vocal ligament and the elastic cone, the upper surface is bounded by the labium vocale and the entrance to the ventricle of the larynx and the lateral surface is bounded by, and is in contact with, the thyro-arytenoid muscle.

The vocal muscle arises from the inferior part of the angular depression between the two laminae of the thyroid cartilage and also from the corresponding vocal fold. Then the fibers run dorsally to be inserted into the lateral aspect of the vocal process and the depression on the ventrolateral surface of the arytenoid cartilage.

The mucous membrane over the vocal folds is stratified squamous epithelium, while below and immediately above, including the ventricle, it is lined with ciliated columnar epithelium. Acinous glands are plentifully found in the larynx except over the vocal folds where they are absent.

This portion of the anatomy of the larynx is presented because it has a direct bearing on the end results, the surgical removal and the treatment of benign lesions.

Since all of the lesions under discussion are confined principally to the vocal cords, the one constant symptom is hoarseness. Laryngeal obstruction is a rather infrequent symptom but does occur occasionally. Pain due to strain of the intrinsic muscles of the larynx may be present.

Papillomatosis in children is probably one of the most difficult types of benign lesions to treat.[2] They are invariably multiple and not only are they found on the vocal cords but elsewhere in the larynx, especially the anterior commissure. These growths, while they do not metastasize, recur locally after removal. Several cases of fatalities have been reported due to asphyxia.

Jackson and Jackson[3] report that they saw 200 cases in their clinics and that the disease is self-limiting but that the masses must be repeatedly removed or scalped off frequently.

Broyles[4] reported five cases treated locally with estrogenic harmone preparation (amniotin in oil) with good results. The results have been questioned on the grounds that since the treatment was carried out over a period of weeks, the recovery may have been spontaneous.

Foster[5] reported good results with small fractional doses of x-ray over the larynx and had no bad results or damage to the larynx. New[6] and Crowe[7] reported the use of radium both externally and internally with good results. Cases of damage to the cartilages and growth of the larynx have been reported by others. The careful repeated scalping of these tumors is still the generally accepted treatment.

Papilloma of the vocal cord in adults is the most common benign lesion. They are found almost always on the free edge of the cord, are usually singular although occasionally they may be multiple. Their frequent location is on the anterior third of either vocal cord. They usually occur in young individuals. Surgical removal by scalping them off without injury to the normal tissue is the best method.

Vocal nodules may be unilateral but are nearly always bilateral. They are always located at the junction of the anterior and middle third of the cord.[8] They are greyish white in color and the surface is smooth. Frequently at the base is a mild area of inflammation or edema. Since they are found in individuals who nearly always abuse the voice, vocal rest will sometimes cause the swelling to subside or completely disappear. More often, however, after the infiltration of fibroblasts has occurred, scalping them off is necessary. Tucker in discussing Jackson's[8] paper on vocal nodules believes that both should be removed at the same time, thus eliminating the vicious cycle produced by these nodules. Cody in discussing the same paper believes that hypothyroidism may affect the larynx in such a way that it is difficult to differentiate the condition from vocal nodules.

Great care must be exercised in scalping them because damage to the normal tissue may result in a permanently damaged voice. For complete healing vocal rest is essential.

Contact ulcers[9] are superficial nonspecific ulcers occurring over the tip of the vocal process on one or both sides. The exact cause

is not known but it may accompany acute infections, especially when there is unusual bumping together of the vocal processes of the arytenoids. While vocal rest will often cure the condition, it may be necessary to remove granulation tissue to allow epithelium to grow over the base and result in healing. The granulation tissue should not be removed deeply.

Fibroid, polypoid, varicose and hyaline tumors are various stages Ash[10] describes under the laryngeal node. He reports the histopathological study of 138 cases collected at the Army Museum. In practically all his cases there was a history of misuse or excess use of the voice which would further substantiate Jackson's conclusion that fibromas are organizing subepithelial hematomas. The tumors almost invariably occur on the free margin of the cord, most frequently on the anterior third. Clinically the four types may be recognized. The fibroid is a low, rather firm, pale, not so sharply demarcated node; the polypoid is a more sharply demarcated, more or less pedunculated, translucent mass; the varicose is more colored from blood content and the hyaline node is pale, quite firm, at times hard. The tumors are removed surgically without injury to normal tissue.

Keratosis may, and frequently does, occur on the vocal cords. New and Erich[11] believe that it may be a precancerous lesion and report squamous cell carcinoma, grade II, in situ, in two of their ten cases of keratosis. Jackson and Jackson report no such incidence either occurring with or following removal.

The process may be considered as a precancerous disease.[12] It is a localized cornification of the epithelium without invasion of the submucosa. With these facts in mind it should not be removed deeply but should be observed indefinitely.

Leukoplakia is considered a precancerous lesion elsewhere in the body and does not differ in the larynx. Not only does it occur on one vocal cord but frequently on both in fairly large areas. Pathologically there is great increase in the horny layers known as hyperkeratosis. This gives the whitish appearance which is seen clinically. The epithelial pegs are greatly exaggerated but the basement membrane is not broken through. Great care and judgment must be exercised in removing tissue for pathological study. After removal over-use of the voice must be prohibited as well as the use of tobacco and alcohol.

Pachydermia is a peculiar mucosal thickening of the epithelium occurring over the anterior surfaces of the arytenoids and adjacent

part of the vocal cords. The lesion is bilateral and symmetrical. Jackson believes that the chief cause is the vasodilating effect of alcohol becoming permanent in the constant drinker. In contradistinction to most other lesions a sharp biting forcep must be used but great care must be taken not to remove normal tissue.

Angioma may be divided into adult and infantile types. The former usually occurs on the edge of the cord while the latter is either subglottic or above the glottis. They are rare lesions and are recognized by their bluish or purplish color. Removal is by direct approach with cupped forceps.

Hematoma when it occurs should be ruptured so that organization of the blood clot will not take place.

Amyloid tumors, cystoma, myoma, myxoma, thyroid tumors, teratoma and lipoma are not often confined to the true vocal cords and are relatively rare. They should be removed without sacrificing normal tissue when they do occur on the true cord.

The ultimate aim in treatment of benign lesions of the vocal cords is to assure the patient of its innocence and to restore the voice to normal. A piece of tissue should never be removed from the larynx without its being sent to the pathologist, regardless of how benign it may appear clinically.[13] With the exception of lipoma and papilloma in children, benign growths manifest little, if any, tendency to recur, therefore, there is no need for radical removal. They do not invade the normal subepithelial layer and this should not be damaged.

From our review of the anatomy of the vocal cord one can see that a deep bite into the cord will result in permanent damage to the elastic tissue, the vocal ligament, or the underlying muscle, with resulting scar tissue and bowing of the cord.

Indirect laryngoscopy for the removal of benign tumors is condemned. The method is too inaccurate and the technique too hard to master.

The personal equation enters into exposing the larynx for an operative procedure. Both the suspension laryngeal speculum as devised by Lynch and the anterior commissure laryngoscope as devised by C. L. Jackson serve well in the hands of various laryngologists. There are some advantages and disadvantages to both. Personally I favor the anterior commissure laryngoscope. It has the advantage that the procedure may be carried out under local or general anesthesia, although I have found general anesthesia rarely

necessary. The ordinary laryngoscope does not serve the purpose for operating on the vocal cords because of its blunt tip. With the anterior commissure laryngoscope one can explore the subglottic region, the ventricular band can be pushed aside so that the ventricle can be exposed and the cord fixed for precise removal of the growth.

A choice of forceps has its advantage. The straight and angulated cup forceps have no substitutes. The edges come together sharply so that precision may be exercised. Occasionally the shearing action of tissue forceps are necessary to remove tough fibrous growths. These, however, are less accurate and should not be used in removing the average benign growth.

A large sized cup forcep should not be used for removing vocal nodules, neither should a small cup be used for large growths. Using the appropriate size makes the removal more accurate.

Stripping the vocal cord when using the cup is one of the disadvantages. This may be eliminated by pushing downward after the tumor is grasped until the upper margin is free and then pulling upward with a rotating motion. According to Lore,[14] however, stripping of the vocal cord has not been a disadvantage as has been thought for years by many noted laryngologists. He reports 100 cases in which he has carried out this procedure with very definitely good results.

The stripping technique is best carried out with the Imperatori subglottic forceps, right and left. The forceps engage the pathological tissue and some of the adjacent tissue. If too much normal tissue is engaged, the forceps' grasp may be relaxed and drawn toward the opposite side until the correct amount of tissue is grasped. The growth is then stripped from the cord. This leaves a straight margin to the cord and not a notched one. The treatment of extensive polypoid degeneration of both cords and chronic hypertrophic laryngitis are best suited to this technique.

Hemorrhage has not been encountered in the removal of benign growths.

The use of the actual cautery or surgical diathermy[15] is condemned in removing benign growths. The destruction of too much normal tissue is likely to take place.

Postoperative care is essentially limited to observation. Early and more satisfactory healing will take place if the patient is placed on a strict regeime of silence until complete healing has taken place.

SUMMARY

1. A brief review of the anatomy of the true vocal cord is submitted so as to show how deep removal of benign growths will permanently damage the voice and throw extra strain on the thyro-arytenoid muscle.

2. Various benign lesions of the vocal cords are reviewed with respect to the latest accepted treatment.

3. Growths of the vocal cords though clinically benign should always be removed and submitted to the pathologist before they are considered to be innocent.

4. Emphasis is placed on the careful removal of benign growths with appropriate instruments and without injury to normal tissue.

5. Except in a few instances a normal voice should be expected after removal.

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REFERENCES

1. Cunningham, D. J.: Anatomy, 5th ed., New York, William Wood and Co., 1930, page 1070.

2. Ferguson, C. F., and Scott, H. W., Jr.: Papillomatosis of the Larynx in Childhood, New England J. Med. 230:477-482, 1944.

3. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Co., 1937, p. 329.

4. Broyles, E. N.: Papilloma of the Larynx, Trans. Amer. Laryng. Assn., 1941, pp. 194-199.

5. Foster, J. H.: Papillomatosis Laryngis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 42:548-559, 1933.

6. New, G. B.: Treatment of Multiple Papillomas of the Larynx in Children, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 30:631, 1921.

7. Crowe, S. J., and Breitstein, M. L.: Papillomas of the Larynx in Children. Report of Eleven Cases, Arch. Surg. 4:275-299, 1922.

8. Jackson, C. L.: Vocal Nodules, Trans. Amer. Laryng. Assn., 1941, pp. 184-193.

9. Thomas, W. C.: Contact Ulcers of the Larynx, West Virginia M. J., June, 1938, pp. 256-260.

10. Ash, J. E.: The Laryngeal (Vocal Cord) Node, Trans. Amer. Acad. Ophth. and Otolaryng., May-June, 1944, pp. 323-332.

11. New, G., and Erich, J. B.: Benign Tumor of the Larynx. Study of Seven Hundred and Twenty-Two Cases, Trans. Sect. Laryng., Otol. and Rhin., A.M.A., 1938, p. 96.

12. Graham, H. B.: Keratosis of the Larynx, Arch. Otolaryng. 36:735-739 (Nov.) 1942.

13. Tucker, Gabriel: Tumors of the True Vocal Cords, Trans. Sect. Laryng., Otol. and Rhin., A.M.A., 1937.

14. Lore, J. M.: Stripping of the Vocal Cords, Arch. Otolaryng. 42:378-384, 1945.

15. Jackson, Chevalier: Treatment of Benign Tumors and Tumor-Like Conditions of the Larynx, Trans. Amer. Laryng. Assn., 1937, pp. 45-48.

XVI

MORTALITY IN CANCER OF THE LARYNX

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In the tumor clinics we are largely concerned with the diagnosis and treatment of cancer. Cancer is not always a fatal disease. Its mortality rate is largely determined by the promptness with which treatment follows the onset of symptoms. If symptoms have been present for three months or less, the prognosis is relatively good. If they have been present for over four months, the prognosis is poor. It is fortunate that many malignant lesions produce early symptoms and are accessible to examination. This is particularly true in the larynx, which is accessible for examination to the general practitioner having only a head mirror and a laryngeal mirror.

Although cancer of the larynx early produces an alteration of voice that is evident to the patient, the number of cases made inoperable through delay is appallingly high. Each survey indicates an increase in the number of five-year cures following surgery. Statistical reports of cures by surgical methods are misleading. About 55 per cent of cases undergoing total laryngectomy are "cured." In 1934, Jackson reported cures in 82 per cent of cases suitable for laryngofissure. The salvage rate for laryngeal cancer appears to be excellent when considered on the basis of operated cases. The question to be answered is: How many cases of laryngeal cancer can be cured of the total number of cases so diagnosed?

From 1930 to 1946 in the Department of Otolaryngology at the University of Illinois a total of 161 cases were diagnosed as cancer of the larynx. Only 40 of these were operated on. Total laryngectomies were performed on 28 patients and laryngofissures on 12 patients. Because we are including cases undergoing operation less than five years ago, we can only speak of them in terms of cure expectancy. On the basis of the above statistics, we may expect 82 per cent of the 12 patients having laryngofissures to be cured; that is, approximately 9.84 people in this group can be expected to survive for five or more years. Again on the basis of the above statistics,

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we may expect 55 per cent of the 28 patients having laryngectomies to be cured; that is, 15.4 of this group can be expected to survive for five or more years. We have then, 25.24 expected "cures." In other words, about one person in 6 1/3, or 15.67 per cent, who develop laryngeal cancer will be "cured."

Since the symptoms of laryngeal cancer manifest themselves early, why were only slightly more than 25 out of 161 curable? Why were only 40 cases considered operable? One patient did not have a laryngectomy because other constitutional factors made him too poor a risk for surgical intervention. Two refused surgery. The remainder had extension of the primary focus, with and without metastases, to areas beyond the reach of surgery.

We must examine the histories of this large incurable group to determine whether inoperability was due to tumor activity, patient neglect, or physician neglect. Whether the carcinoma of the larynx is of squamous or cuboidal cell origin, it is usually a relatively slow process and inoperability can not, except in unusual cases, be ascribed to tumor activity. Most cases are incurable because of procrastination which must be laid to the patient or to the physician.

The patients as seen at the University of Illinois are predominantly male; the average age is 60.83 (29-83) years. They have been aware of a change in voice for an average of 9.46 months. Only 27 of our patients (16.77 per cent) had symptoms for less than four months. Forty-five per cent have seen no physician prior to appearance in our clinic. Thirty per cent were referred to us by the only physician visited. In one instance, the physician referred the patient to us after having treated him with less than cancerocidal radiation therapy. In 25 per cent of the cases the patient was variously treated by two to five physicians for a variety of conditions. Diagnoses included laryngitis, bronchitis, pharyngitis, sinusitis, common cold, asthma, and hypertension. Therapy included nasal douches, sinus irrigations, swabbing of the oropharynx, cautery to the pharynx, penicillin lozenges, penicillin-aerosol inhalations, gargles, cough syrups, and in one case, faith healing. Of the 40 patients in this group, only three are alive after one, one and one-half and two years, respectively, since admission to our clinic.

From the above we can draw certain inferences. In 45 per cent of all the cases, patient procrastination, and in 25 per cent medical mismanagement was responsible for the delay. In 1.86 per cent of the cases, surgery was refused or was contraindicated because of other

conditions. In 11.37 per cent of the cases, the delay was due to combined patient and physician procrastination. When we examine our cure expectancy, 15.67 per cent, we find that it approximates the percentage of patients having symptoms for less than four months, 16.77 per cent of the total number of cases seen.

While it is true that in most conditions seen by physicians, recovery will occur regardless of therapy, this does not hold true for cancer. The current practice of treating every complaint with antibiotic and chemotherapeutic agents without making a diagnosis is most reprehensible. It is apparent, as shown by Leach and Robbins, that the physician unfortunately has not been made sufficiently aware of the prevalence of cancer or conscious of the importance of the condition in the diagnostic picture. In every age group the possibility of cancer must be excluded before the nonfatal disease entities are considered.

How prevalent is cancer of the larynx? While the larynx is the primary site of cancer in only 0.9 per cent of all cases, it constitutes 19.1 per cent of the malignancies of the head and neck. While cancer of the larynx occurs comparatively infrequently, it has very early symptoms and is easily accessible to examination and biopsy. Not all hoarseness is due to cancer. A number of conditions in the respiratory system are associated with hoarseness. These, however, with the exception of miliary tuberculosis, have a negligible mortality rate and should not be considered until cancer is excluded.

We reviewed the laryngeal biopsies performed in the Department of Otolaryngology at the University of Illinois to determine the part played by cancer in the picture of chronic laryngitis. Malignancies constituted 55.82 per cent of the total laryngeal biopsies. The diagnoses of the benign 44.18 per cent included polyps, polypoid thickening of the vocal cord, papilloma, leukoplakia, tuberculosis, and blastomycosis. How many of these would eventually show malignant changes is impossible to determine. It is our feeling that the leukoplakias are definitely suspect. None the less, we find that almost half of the cases of chronic laryngitis are benign conditions.

The low incidence of cancer of the larynx and the fact that about half of the cases of chronic laryngitis are benign tend to lull both the patient and the physician into a false state of security. When these facts are considered together with the results of laryngeal surgery, they produce an optimism diametrically opposed to the facts. Of 100 persons who develop chronic laryngitis and have a tissue diagnosis about 47 will die of laryngeal cancer.

Statistics on the number of operated cases which were cured were valuable during the years prior to the advent of chemotherapy, antibiotics, modern preoperative preparation, and operative and postoperative supportive measures. They indicated the value of particular techniques and did much to increase general surgical skill. Today concentration upon a high operative-cure rate indicates an increasing restriction of the indications for surgery. Following this trend we may achieve 100 per cent surgical cures, but the mortality rate of the disease will rise accordingly.

Laryngeal surgery has reached a stage where variations in technique add little to the statistical picture. For this reason it is time that a change be made in the point of reference. The emphasis today must be on diagnosis. It is essential that the physician be made more cancer-conscious. Percentage of cures in relation to cases diagnosed may highlight the appalling situation and so reduce the time loss due to physician procrastination.

CONCLUSIONS

1. More than half the cases of chronic laryngitis are cancer and even some of the benign conditions are suspect for eventual malignant metaplasia.

2. Only 15.67 per cent of individuals having cancer of the larynx can be expected to survive five or more years because of patient and physician procrastination.

3. The incidence of 44.18 per cent benign laryngitis and the relatively high incidence of surgical cure of operated cases give the patient and the physician a false sense of security.

4. Education of both the public and the physician to the implications of chronic laryngitis is a requisite to a higher cancer cure incidence.

5. Repeated appraisal of each case of chronic laryngitis by the attending physician will be rewarded by an increased cure rate in cancer.

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Clinical Notes

XVII

PHLEBOLITH OF THE ANTERIOR FACIAL VEIN

CASE REPORT

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Phleboliths are encountered frequently in the veins of the pelvis, prostate, and leg; they occur infrequently in the upper extremities, but rarely in the region of the head and neck.

The pathology of phleboliths is well described by Moore[1]:

> "Within a few days after a thrombus forms, there are proliferations of fibroblasts, invasion by endothelial sprouts and capillaries and beginning organization. The fibrin and dead tissue are removed by autolysis and by phagocytosis. The proliferating endothelial sprouts join with one another and, eventually, continuous channels through the mass of fibrous tissue are established, a process known as canalization of a thrombus. In larger thrombi containing an excess number of leucocytes, death of these cells and liberation of their enzymes may bring about puriform softening of the centers. The superficial layers are protected by antitryptic substance in the blood. Some thrombi, especially those in pelvic veins, undergo organization and final calcification to form phleboliths."

De Vries[2] in 1930 reported a case of orbital phlebolithiasis associated with exophthalmos. In 1932 Gastreich[3] reported a similar case of orbital phlebolithiasis. In 1930 Dieterich[4] reported a case of phlebolith in an angioma of the masseter muscle which was mistaken for a cystic parotid tumor.

A review of the literature fails to reveal a single incidence of phlebolith in the anterior facial vein. Byars[5] had a case in 1947 with gross findings similar to those in the following case.

REPORT OF A CASE

A 37-year-old housewife was admitted to McMillan Hospital on August 13, 1946, complaining of recurrent episodes of pain and

Fig. 1.—Left lateral view of mandible showing spheroid calcifications at the angle of the jaw.

more or less constant swelling of the left face and jaw. The onset of swelling, which was first noticed sometime after recovery from typhoid fever at the age of six, was especially noticeable when lying down at night or when engaged in strenuous exercise or housework. There were periods of weeks or months when there was constant swelling associated with pain and low-grade fever. In 1935 the area, which was quite large and tender at that time, was incised with considerable bleeding which continued for three days. No pus was found and no improvement followed. In 1942 she was told she had salivary stones, and an attempt was made to remove the calculi through the floor of her mouth. No stones were found, and severe bleeding continued for four days after surgery. In March 1946, an external incision was made for the purpose of removing salivary calculi. A dark "stone" resembling a dry, firm blood clot was removed, but her symptoms continued unchanged. On August 7, 1946, an x-ray diagnosis of calculi in the left submaxillary region was made by a competent radiologist, who advised operation.

Examination on admission gave no significant findings except a slightly reddened area of swelling over the left side of the face and jaw, extending about 4 cm. forward from the angle and 2 cm. above

and below the lower border of the left mandible. Superficially the skin felt "puffy." On deeper palpation the area was quite tender, firm, and slightly nodular. A horizontal scar was present below the lower border of the mandible. Adjacent nodes were not enlarged. The patient's temperature was not elevated and laboratory findings were within normal limits.

X-ray Findings.—Lateral view of the mandible (Fig. 1): Several small areas of calcification are seen in the soft tissues inferior to the angle of the mandible on the left, which may represent stones in the left submaxillary gland. X-ray diagnosis: Calculi in the submaxillary gland, left.

On August 14, 1946, the patient was taken to the operating room.

Operative Procedure.—Under 2% novocaine infiltration anesthesia the left submaxillary gland was exposed by an incision through the old scar. The gland was freed with difficulty because of fibrosis in the surrounding tissues. Careful palpation revealed no evidence of stones in the gland or the ducts. Because of extensive mobilization, the duct was followed to the floor of the mouth, ligated, and the gland removed. Further dissection and elevation of the upper flap of the wound revealed what appeared to be a large irregular venous space in the course of the anterior facial vein, roughly 5 by 3 cm. when completely exposed. Lying within this space and extending forward from the anterior border of the masseter muscle, were several round, hard bodies from 6 to 8 mm. in diameter. What appeared to be a fairly recent large thrombus was evacuated from the posterior portion of the sac. After ligature of the vessel above and below the area exposed, the sac and the calculi, which were enmeshed in a fibrous network within the sac, were freed from the underlying periosteum and fascia and removed in one piece. Bleeding from the small branches of the external maxillary artery necessitated ligature of that vessel. The parotid duct was not exposed and subsequent observation showed no disturbance in function of the parotid gland. The cervical fascia and platysma were closed with 00 catgut. A narrow rubber dam drain was placed deep in the cavity and the skin closed with interrupted silk sutures.

Pathological Report.—"The specimen consists of a number of round, firm bodies varying from 2 to 5 mm. in diameter embedded in fibrous tissue. Between these structures a dilated duct can be traced. On section the bodies show a laminated structure and the majority of them are calcified. Hematoxylin and eosin-stained sec-

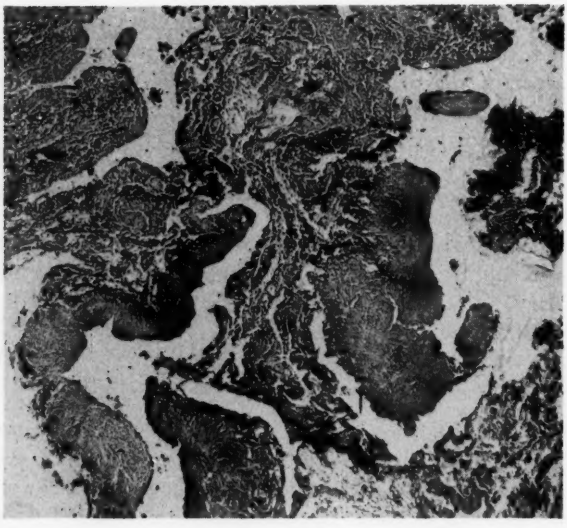

Fig. 2.—Photomicrograph of section showing venous spaces.

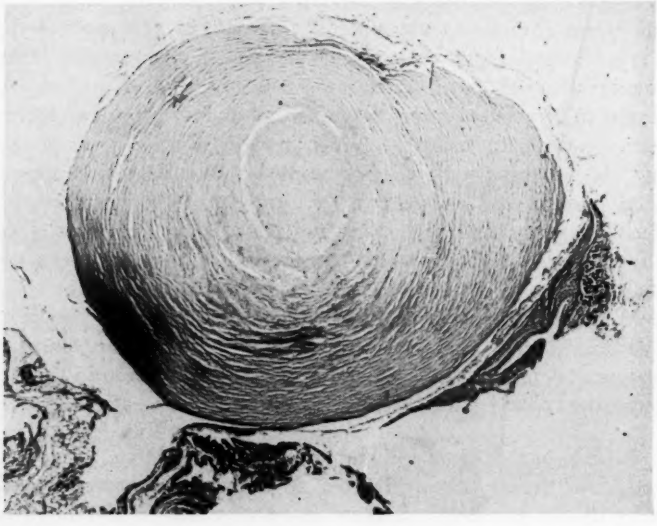

Fig. 3.—Photomicrograph of section of lamellated phlebolith, greatly magnified.

tion shows a spheroid body (Fig. 3) which is lamellated in character. The outer portions consist of dense, acidophilic, relatively acellular, concentrically arranged island connective tissue fibers. Rarely a thin-walled capillary is seen. The central portion of the section, though poorly delineated from the outer hyalinized fibrous material, is continuous with and gradually assumes a more loose though acellular acidophilic character. A few tiny blue stippled areas, resembling calcium, are seen. Elastic tissue stain fails to reveal the presence of any elastic fibers. A few tags of fibrovascular material are adjacent to the periphery of the spheroid body. A section through fibrovascular tissue (Fig. 2) reveals a few small blood vessels and one moderately large, relatively thick-walled structure, void of blood and resembling in character the wall of a vascular channel. The wall of this structure is composed of mixed fibrous, muscular, and elastic material as demonstrated by the Verhoeff Van Gieson stain. Diagnosis: Fibrovascular tissue and hyaline body consistent in appearance to that of a phlebolith."

Following the operation there was serosanguineous drainage and moderate edema of the face, which continued for several days. The wound was completely closed in two weeks, and the patient reported after six weeks that she was symptom free for the first time in her memory. She continued improved until a year after operation when she noticed a small nodule in the left cheek. Examination revealed a small shot-like node just palpable on the buccal surface of the cheek. X-ray films of the area failed to show any evidence of calcification and the mass was assumed to be a small lymph node. No further symptoms have been reported.

SUMMARY

1. A case of phlebolithiasis of the anterior facial vein is presented.

2. Phleboliths must be considered in the differential diagnosis of salivary calculi of the submaxillary glands.

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REFERENCES

1. Moore, R. A.: Textbook of Pathology, 1st ed., Philadelphia, W. B. Saunders Co., 1944, p. 99.

2. de Vries, S.: Angioma carvernosum der orbita von 64 Jahrigem bestand mit röntgenologisch nachweisbaren phlebolithen., Klinische Monatsblatter Für Augenheilkunde, 85:538-548 (July-Dec.) 1930.

3. Gastreich, C.: Phlebolithen bei orbitalvarizen mit intermittierendem exophthalmus, Klinische Monatsblatter Für Augenheilkunde, 88:773-777 (Jan.-June) 1932.

4. Dieterich, H.: Zum auftreten vom phlebolithen an der wange. Duetsche Zeitschrift für Chirurgie, 229:389-390 (Dec.) 1930.

5. Byars, Louis: Personal communication.

XVIII

ACUTE PYOGENIC MENINGITIS FOLLOWING TONSILLECTOMY, WITH RECOVERY

A CASE REPORT

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A case of acute pyogenic meningitis following tonsillectomy was encountered in the course of otolaryngologic practice. The rarity of such a complication or sequela of tonsillectomy, combined with other interesting features of the case, warrants the following presentation. This may be particularly opportune in view of the controversial "poliomyelitis season" with its inevitable and varied opinions regarding the advisability of performing elective surgery of the head and neck. A brief résumé of the literature and the case report are hereby submitted.

Of all the intracranial complications in otolaryngeal regional fields, those arising from infections in the tonsillar fossae are the rarest, next to those from the larynx and the esophagus. Leatherwood[1] reported only one case of purulent meningitis, with recovery, in a series of 10,000 tonsillectomies. Nesbitt[2] reported a fatal case of meningitis among 1,457 tonsillectomies. Loeb,[3] in a review of 332 fatalities following nose and throat operations performed throughout the United States, reported four cases of post-tonsillectomy meningitis and one case of post-adenoidectomy meningitis. Chiavis' clinic reported 6,000 tonsillectomies without a single intracranial complication.[4]

In a survey of the literature (1900-1940), Hara and Courville[5] found a total of 76 cases of intracranial complications of tonsillar origin, nine of these being classified under the acute primary meningitides. Only 28 cases of fatal intracranial or spinal lesions were reported among the great number of tooth extractions performed on Army personnel.[6]

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Intracranial lesions of tonsillar origin may be noninfectious or infectious in nature.[5]

The noninfectious type may arise from embolic occlusion of cerebral vessels with subsequent softening of brain tissue, rupture of a cerebral blood vessel, or anesthesia accidents (status asphyxia and sensitization due to operative manipulation of the tonsillar region). The use of local anesthesia and general narcosis as important etiological factors are stressed by Jacobsen.[4]

However, the great majority of intracranial complications are caused by the extension of the infection by some route from the tonsillar fossa to the brain. Hara and Courville[5] discuss in detail the four chief pathways by which infections can be transmitted from the pharynx to the intracranial structures. One or any combination of these invasion routes may be involved in producing the resultant lesion:

1. By way of the perineural sheaths of the extracranial nerves and the lymph channels, the infections being carried directly into the subarachnoid system. This mode of extension is rare.

2. By way of the tissue spaces. The tonsillar fossa is separated from the surgically significant parapharyngeal space by the superior constrictor muscle and its fascia. The styloid process divides the prestyloid compartment (pharyngomaxillary fossa), which is intimately connected with the tonsillar fossa, from the poststyloid compartment, which contains the great cervical vessels and nerves. Infections may extend directly along the parapharyngeal space and enter the cranium by congenital dehiscences, by acute inflammatory erosions in the bony wall, and by haversian canal venules in the cranial bones. Experimentally, Wessely[4] demonstrated that fluid injected into the tonsillar regions of a cadaver could be recovered at the base of the cranium, apparently by extension through the parapharyngeal space.

3. By way of the arterial channels, namely, the internal carotid and vertebral arteries, which supply the skull and its contents. This leads to the production of meningitis, multiple abscesses, or thrombosis. The extremely rich blood supply of the tonsillar regions, derived from the close proximity of the external and internal carotid arteries, is instrumental in enabling infections to travel towards the cranium by way of the perivascular structure (as reported in two cases of acute meningitis following severe tonsillitis[5]) and by way of the arterial stream itself (as the result of a needle punc-

ture of the wall of the carotid artery incident to the induction of local anesthesia[7]).

4. By way of the venous channels, with the production of a thrombosis of the peritonsillar vein. This has been reported following tonsillectomy, the result being an extension of a retrograde thrombophlebitis.[8] In cases of tonsillitis with venous thrombosis the infection may enter the cranium either (1) by mesial extension of the thrombus in the basilar vein and the pterygoid plexus, by way of the veins of Versalius through the foramina ovale and lacerum, the sphenoid bone, and a communicating branch of the ophthalmic vein, to reach the cavernous sinus; or (2) by lateral extension of the thrombus by way of the internal jugular vein to the lateral sinus and the torcular Herophili, thereby reaching the intracranial blood spaces.

Dixon and Helwig,[9] in a study of six cases, believe that the thrombophlebitis of the internal jugular vein occurring after acute tonsillitis is similar in every way to the septic thrombi in the other veins, as in the sigmoid sinus in mastoiditis. The phlebitis usually is limited to the immediate vicinity of the primary site of the infection. They believe that actual invasion of the cerebral tissues must take place by another route.

Brunner[10] states that lymphangitis and lymphadenitis always accompany an acute tonsillitis or peritonsillitis. According to him the infection is then transmitted from the perivenous lymph channels to the venous channels with or without the formation of a thrombus.

Jacobsen believes that streptococci and various other microorganisms existing in the tonsillar crypts are liberated during the process of removing the tonsils. Elliott[11] recovered hemolytic streptococci from the tonsils of 87 out of 137 patients undergoing tonsillectomy (64 per cent). These bacteria are pressed through the surfaces of the denuded areas into the lymphatics, thus causing an apparent sepsis.

Infections of the meninges originating in the pharynx, as seen in acute tonsillitis or after tonsillectomy, or appearing during or after various otogenic and rhinogenic conditions, may occur in two ways: (1) a phlegmon of the parapharyngeal space may reach the skull and primarily infect the meninges by direct extension, and then produce intradural suppuration and thrombophlebitis of the cavernous sinus and carotid plexus, or (2) the infection may travel by way of the

blood vessels, with the meninges as the primary intracranial structure involved. In some cases the brain itself is infected first, while the meninges either escape infection entirely or are involved secondarily, as is seen, for example, with thrombophlebitis of the cerebral sinuses or with extension from an osteomyelitic brain abscess.

Bacteremia is relatively frequent following tonsillectomy, tooth extraction and similar operative procedures. Microorganisms can usually be cultured from the blood stream anywhere from a few minutes to two or three hours postoperatively. In all but exceptional instances the bacteremia is inconsequential and of no significance. According to Haymaker a streptococcus viridans is the usual organism cultured. Brunner states that various types of streptococci and staphylococci have been isolated, with hemolytic streptococci being found only on rare occasions. Elliott reported that a transient bacteremia was observed in 38 out of 100 patients within a few minutes after tonsillectomy, with the isolation of Streptococcus pyogenes, Streptococcus viridans, Streptococcus pneumoniae, Haemophilus influenzae, staphylococcus, and corynebacterium.

Some authors believe that there is some connection between tonsillectomy and poliomyelitis. There is some evidence that certain virus infections, as influenza, measles, and particularly poliomyelitis, are facilitated and influenced by tonsillectomy. In these cases, it is assumed that the virus travels along the nerve pathways, particularly over the third division (mandibular branch) of the trigeminal nerve, with no involvement of the parapharyngeal space.

The incidence of intracranial virus infections following tonsillectomy is very low. In a series of several thousand tonsillectomies performed during the past 25 years in his clinic, Brunner[12] noted only two instances of intracranial virus infections. Page[13] reported only one case of poliomyelitis following 8,915 tonsillectomies.

However, Krill and Toomey[14] presented a study on the occurrence of bulbar poliomyelitis in its worst form in five out of a family of six children. Tonsillectomies and adenoidectomies had been performed previously on the five children. Similarly, Anderson[15] found the incidence of poliomyelitis in children having recent tonsillectomies 2.6 times greater than in the general child population.

With reference to the cases of Krill and Toomey, McCormick[16] recently discussed the possible etiological relationship of the basic lymphatic involvement back of the operative procedure and the effect of the surgical anesthetic from the viewpoints of nutritional

deficiency and anoxia. He suggested that the virus may actually be an endogenous biochemical by-product of disturbed carbohydrate metabolism (vitamin B_1 deficiency), capable of producing a similar disease by catalytic chemical action on the central nervous system.

REPORT OF A CASE

D. T., Jr., a 21-year-old, single, white male, was admitted on May 21, 1947, to the Department of Otolaryngology, Veterans Administration Hospital, Hines, Illinois, with the diagnosis of "chronic tonsillitis." He gave a history of having had his first acute attack of tonsillitis in August 1944, while on duty with the U. S. Navy as a radar operator, which necessitated hospitalization for one week. Since that time he has had a number of similar mild attacks of "sore throat", lasting only two days as a rule and clearing up with salt water gargles. These episodes, which were not associated with any constitutional symptoms, dysphagia, or fever, recurred frequently during the winter of 1944-45; relatively few attacks were apparent during the years 1945 and 1946. The last attack, which occurred in December 1946, also subsided after two days. The patient had no complaints of any kind in the six months preceding hospitalization. He requested admission for a tonsillectomy, which had been recommended to him previously. Except for chickenpox in childhood, the patient's history was completely negative.

The familial history was not significant. Following his discharge from the U. S. Navy in May 1946, the patient lived with his family in Monongahela, Pennsylvania, until September 1946, when he moved to Chicago, Illinois. Prior to his admission to this hospital, he lived with a friend and was employed by a large mail-order company. Careful questioning of the patient failed to reveal any contact with, or knowledge of, any illnesses among his associates at work or among his acquaintances.

Upon admission, the patient had no complaints. Physical examination was essentially negative except for hyperplastic tonsils of moderate size. The mouth and pharynx otherwise appeared to be normal. The blood serology was negative, and normal coagulation and bleeding times were noted. On May 26, 1947, the day of operation, a re-examination of the patient failed to reveal any new findings. Conventional preoperative medication was administered (barbiturate orally and morphine gr. ¼-atropine gr. 1/150 by "hypo"). The local anesthetic consisted of 1.5% procaine with 1:1000 adrenalin hydrochloride. The tonsillectomy was performed without difficulty.

The postoperative course proceeded uneventfully until 26 hours later, when the patient began to complain of generalized headaches. The headaches became worse and within a few hours were accompanied by nausea and projectile emesis. The temperature was 104° F., pulse 115, respirations 23. He was examined by the duty medical officer (30 hours postoperatively), who found no evidence of neck rigidity or any abnormal neurologic signs. The patient was given 40,000 units of penicillin every three hours intramuscularly and one liter of 10% glucose intravenously.

The symptoms grew progressively more severe and re-examination 44 hours postoperatively revealed an acutely ill patient with marked stiffness of the neck and hyperactive reflexes of both lower extremities. There was a suggestive Babinski sign on the right and Kernig's and Brudzinski's signs were positive. The temperature was 102°, pulse 115, respirations 23. The patient was seen by the medical service and a diagnosis of meningitis was made.

At this time, the following laboratory data were noted; chest roentgenogram negative; urine negative; 4,700,000 erythrocytes per cu. mm., 13.5 grams hemoglobin; 20,200 leucocytes per cu. mm., 97% polymorphonuclears, 3% lymphocytes.

A spinal puncture was performed and turbid spinal fluid was obtained under increased pressure (230 mm. by water manometer). Cells numbered 1296 with 88% polymorphonuclears and 12% lymphocytes; the globulin content was increased, the Pandy test was two plus. Repeated smears from the spinal fluid failed to reveal any organisms. Cultures were negative after ten days. The Levinson and Wassermann tests were negative. The Lange gold curve was not significant, 0000012210.

The patient was immediately isolated and given large doses of sulfadiazine orally and penicillin intramuscularly. For two days no apparent change was noted in the patient's condition. Then the patient began to show gradual improvement. The headaches and meningeal signs slowly abated and finally subsided completely six days after the initiation of intensive therapy.

During this period, various diagnostic procedures were performed. The tuberculin test (0.1 cc. first strength purified protein derivative intradermally) was negative after 48 hours. The Schick and Dick tests were negative. Blood and spinal fluid smears and cultures were consistently negative. Nose and throat cultures revealed hemolytic staphylococcus aureus predominantly with occa-

sional gram-positive cocci and diphtheroid organisms. The coagulin test was negative. Blood chemistry constituents were within normal limits.

The convalescence was uneventful and the patient was discharged as cured 23 days after the onset of the meningeal complications.

Comment.—This case is being presented for theoretical considerations primarily; practically, our epidemiological and bacteriological investigations were inadequate for a number of reasons. It is conjectural whether the 200,000 units of penicillin administered prior to lumbar puncture may have been responsible for the negative results obtained on repeated smears and cultures of blood, spinal fluid, and nose and throat specimens. The hospital pathologist felt that the organisms isolated in the cultured nose and throat specimens were contaminants. The early lumbar puncture or the previous administration of penicillin, or both, may have been responsible for the findings of an acute pyogenic meningitis instead of a frank purulent meningitis. Brunner[17] states that in the majority of cases the spinal fluid is cloudy and organisms are present; however, cases have been seen in which no bacteria could be found in the cerebrospinal fluid up to the time of death of the patient, but meningitis was found at autopsy. A thorough pathological examination of the tonsils would have been most informative; unfortunately, the tonsils were destroyed by the time the complication became apparent. Therefore, this case is to be considered primarily as a clinical contribution to the problem of posttonsillectomy complications.

Firstly, we must consider the possibility of a coincidental meningococcal meningitis. An interesting history is presented of frequent episodes of a characteristic mild type of "sore throat." There is some evidence that catarrhal disorders of the nose and throat predispose to this type of infection.[18] The patient may have been infected through contact with a carrier or he may have been a carrier himself for a period of time before developing the disease. As far as could be determined, there was no history of, or contact with, any cases of meningitis. No cases of meningococcal meningitis were reported locally during this period. We know that meningococci can be demonstrated in the spinal fluid in 90 to 100 per cent of cases of epidemic meningitis; thus, if this were such a case, the specific organisms would undoubtedly have been isolated, in spite of the previous penicillin therapy. The excellent response to penicillin-sulfonamide therapy is in itself not significant since other types of meningitis react equally well.

There is no reason to consider seriously the possibility of a poliomyelitis or other virus infection in view of the clinical course, laboratory findings, and therapeutic response of this case.

It is entirely possible that the patient may have been a "healthy carrier" of some dormant pathogenic microorganism, which needed only an "opening" or a breakdown of the local protective tissues in order to induce a fulminating infection. Against this premise is the strikingly short interval (26 hours) between the operation and the onset of the prodromal symptoms of the meningeal complications. This is not compatable with the average incubation period required for the usual potential pathogens of the nose and throat passages to enter the traumatized areas and produce disease.

The extremely short interval was a most significant factor in speculating on the possible route or routes for transmission of the infection in this case. Hara and Courville reported a case of postadenoidectomy meningitis, resulting in death two weeks postoperatively. In Jacobsen's case of posttonsillectomy meningitis the patient developed signs and symptoms of the complications four weeks postoperatively. Nesbitt's patient died of a meningitis 14 days after operation. Haymaker reported on two patients with leptomeningitis who died 5 days and 27 days postoperatively. The patient of Cadbury and Siddall died of a cerebral abscess five days after a tonsillectomy.

In view of this time factor, the following was concluded regarding the pathogenesis of the intracranial complication: either (1) the infection reached the meninges as a result of a postoperative bacteremia, or (2) the infection was caused directly by a needle puncture of the wall of the carotid artery during the induction of local anesthesia. Of course, only theoretical conclusions can be drawn, but it is most probable that the meningitis was the direct aftermath of the postoperative bacteremia.

Earlier in this paper mention is made of the various organisms usually isolated in cases of postoperative bacteremia, namely, various types of streptococci and staphylococci, H. influenzae, and corynebacterium. Any of these organisms may have been concerned in the production of this meningeal complication.

SUMMARY

1. A report on a patient with acute pyogenic meningitis following tonsillectomy has been presented.

2. This meningitis probably was the aftermath of a post-operative bacteremia.

3. The pathogenesis of intracranial complications of tonsillar origin has been reviewed.

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REFERENCES

1. Leatherwood, T. F.: Complications Following Tonsillectomy, J. Tennessee M. A. 21:272 (Nov.) 1928.

2. Nesbitt, B. E.: Post-Operative Complications and Results of Tonsil and Adenoid Operations in Children, Brit. M. J. 2:509 (Sept. 15) 1934.

3. Loeb, H. W.: Fatalities Following Operations upon the Nose and Throat Not Dependent upon Anesthesia—A Study of 332 Hitherto Unreported Cases, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 31:273 (June) 1922.

4. Jacobsen, C.: Complications Attending Operations on Palatal Pharyngeal Tonsils, Acta Oto-Laryng. 10:568-70, 1927.

5. Hara, H. J., and Courville, C. B.: Intracranial Complications of Tonsillar Origin, with Special Reference to Incidence and Pathogenesis, Arch. Otolaryng. 35:530-571 (April) 1942.

6. Haymaker, W.: Fatal Infections of the Central Nervous System and Meninges after Tooth Extraction, with Analysis of 28 Cases, Am. J. Orthodontics (Oral Surgery Sect.) 31:117-188 (March) 1945.

7. Cadbury, W. W., and Siddall, A. C.: Cerebral Abscess Following Tonsillectomy, China M. J. 44:910 (Sept.) 1930.

8. Fetterolf, G., and Fox, H.: The Reaction of the Paratonsillar Tissues to Tonsillectomy: A Study in the Etiology of Post-Tonsillectomy Pulmonary Abscess, Am. J. M. Sc. 166:802 (Dec.) 1923.

9. Dixon, O. J., and Helwig, F. C.: Thrombophlebitis of the Internal Jugular Vein as a Complication of Tonsillitis—Study of Six Cases, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 39:1137 (Dec.) 1930.

10. Brunner, Hans: Infections of the Para-pharyngeal Space, Arch Otolaryng. 31:597 (April) 1940.

11. Elliott, S. D.: Bacteremia Following Tonsillectomy, Lancet 2:589-592 (Sept. 9) 1939.

12. Brunner, Hans: Virus Infections Subsequent to Tonsillectomy, Confinia Neurologica, Fasc. 1/2, (S. Karger) Basel, New York, Vol. VII, p. 13, 1946.

13. Page, J. R.: Tonsillectomy and Poliomyelitis; One Case of Poliomyelitis Following 8,915 Tonsillectomies, Arch. Otolaryng. 39:323-324 (April) 1944.

14. Krill, C. E., and Toomey, J. A.: Multiple Cases of Tonsillectomy and Poliomyelitis, J. A. M. A. 117:1013 (Sept. 20) 1941.

15. Anderson, J. A.: Poliomyelitis and Recent Tonsillectomy, J. Pediatrics 27:68-70 (July) 1945.

16. McCormick, W. J.: Tonsillectomy and Poliomyelitis; New Concept of Etiological Relationship, M. Rec. 156:164-167 (March) 1943.

17. Brunner, Hans: Pathology and Treatment of Otitic and Rhinogenic Meningitis, Illinois M. J. 77:57-63 (Jan.) 1940.

18. Brain, W. R.: Diseases of the Nervous System, Oxford University Press, London, 1933, p. 319.

XIX

SIMULTANEOUS POLIOMYELITIS IN FOUR BROTHERS TWO OF WHOM HAD RECENTLY HAD TONSILLECTOMIES

A. C. Hilding, M.D.

Duluth, Minn.

There still seems to be a great deal of doubt as to the causative relationship between tonsillectomy and bulbar poliomyelitis. It is with this in mind that the following report is made.

The season for poliomyelitis seems to start somewhat later in Duluth than in other parts of the country, and those surgeons who feel that there might be a relation between tonsillectomy and poliomyelitis have thought that the first part of August was early enough to discontinue this operation. On the morning of August 2, 1944, I followed another surgeon into the operating room who remarked, as he came out, "Well I have done my last tonsillectomy until frost."

I performed tonsillectomies that morning on two brothers, A. L. and C. L. Both operations were uneventful and there were no complications during the first two weeks.

One of the boys (A. L.) became ill on August 16 (14 days after tonsillectomy) with general malaise and weakness. His mother called me stating that he had difficulty in speaking. Upon examination it was found that he showed signs of bulbar palsy, and he was readmitted to the hospital on August 17. He had difficulty pronouncing his words, some trouble in swallowing, and the palate seemed weak. There was no headache or stiffness of the neck. Neurologic findings were essentially normal. The spinal fluid test revealed no white blood cells and the Nonne test was negative.

Another spinal fluid specimen was drawn on August 18, however, which contained 100 blood cells, and the proteins were 57 mg. per 100 cc. Sixty-five per cent of the white blood cells were neutrophiles and 35 per cent lymphocytes. A leukocyte count of the blood revealed 15,000 leukocytes, and the hemoglobin was 12.5 grams. A differential blood count showed 93 per cent neutrophiles. The patient's temperature rose to 102° on the 18th, and remained ele-

vated until death. Some stiffness of the neck developed during the 18th and 19th, and on the 19th his pharyngeal paralysis seemed to become almost complete. He was very listless for two days and gradually became stuporous and eventually unconscious. At this time he began to grow cyanotic. He was put in a respirator and given oxygen, and postural drainage was attempted. However, he died on August 23 with symptoms of encephalitis and agonal opisthotonos.

The patient had three brothers, all of whom developed poliomyelitis. One on August 19, one on August 20, and the last on August 21. The brother (C. L.), who had undergone tonsillectomy on the same day as the patient, become very ill and had paralysis of the right foot and leg. He was readmitted to the hospital on August 21 where he stayed until September 10. In addition to the foot drop there was neck rigidity, pain in the thighs, and fever. A specimen of the spinal fluid was drawn and found to contain 173 white blood cells. The third brother (CL. L.) was admitted to the hospital on August 23 complaining of stiff neck, stiff painful back, and trouble in swallowing, and headache. The difficulty in swallowing continued until August 25, after which date there was no further trouble. This boy recovered completely. L. L. (the fourth brother) was admitted to the hospital on August 23 and remained until September 10. He was admitted with complaints of headache, fever, cough, stiffness and soreness of the neck, back, and thighs. His spinal fluid count was 43 cells. After admission to the hospital he developed pain and weakness in the left shoulder, and on September 1 and 2 he had difficulty in swallowing and his food tended to regurgitate through his nose. This boy also recovered completely.

The Health Department of the city investigated the case and found that the neighborhood boys had been swimming in a stagnant pool on a small creek which was contaminated by sewage. One of the playmates (W. M.) became ill about August 1 with fever, vomiting, and headache. Despite his illness he did not stay at home and during this period played with the patient and his brothers. Another playmate (L. J.) living just across the street from the patients became ill with poliomyelitis and paralysis about August 10 or 12. A third playmate (A. J.)—a cousin of the second and living next door—became ill with the general symptoms and was hospitalized. In fact there was a small local epidemic, so definite that when I called the Health Department to report my case the first question asked was, "Does he live around 20th Avenue West?" My patients' home was on 18th Avenue West.

COMMENT

The source of infection in these four cases of poliomyelitis among four brothers seems to be fairly clear: namely, the stagnant pool contaminated by sewage. These four brothers became directly infected from the water or indirectly infected through their playmates.

The unusual circumstances of poliomyelitis in four brothers, two of whom had had tonsillectomies two weeks previously, would seem to furnish an ideal situation for determining a causal relationship between tonsillectomy and bulbar poliomyelitis. However, ignoring the fact that the number is too small to be statistically significant, the issue still remains beclouded. Three of the four developed bulbar symptoms and only one of these had undergone tonsillectomy. The one who had undergone tonsillectomy, however, was very much more ill than the other two and eventually died. However, the second one who had undergone tonsillectomy (C. L.) was the only one of the four who showed no degree of bulbar palsy.

No definite conclusions can be drawn from these four cases. However, in connection with other cases reported in the literature they may be of some significance.

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XX

ATELECTASIS IN THE NEWBORN. CASE REPORT

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WATERVILLE, MAINE

Atelectasis is a serious affliction at any age. It is one of the commonest of postoperative complications and may occur in a variety of conditions predisposing to interference with normal lung expansion. The cause may be mechanical, such as obstruction; biochemical, from altered oxygen and nitrogen proportions in inspired air; vasomotor, from shock; or impaired defense mechanism of the lung, resulting from infection or trauma.

When atelectasis occurs in the newborn the outcome is always critical and may be fatal unless relieved. Obviously the morbidity is affected by the promptness with which therapy is instituted. The most common cause is the aspiration of amniotic fluid coincident with or following delivery, whether normal or by section. There is some evidence to suggest that this occurs somewhat more frequently in infants delivered by cesarean section. This may be because there is a greater natural stimulation to infant respiration in a normal delivery and to the previously ruptured membranes, while the infant delivered by cesarean section is extracted from a sac filled with amniotic fluid. Excessive maternal narcotization may likewise be a factor, resulting in a depression of the infant's respiratory center.

Cyanosis, possibly intermittent at first but gradually increasing, is the predominant sign and should indicate the probability of atelectasis. Shallow respiration, rapid pulse and absence of breath sounds are suggestive. X-ray examination usually confirms the diagnosis, showing a mediastinal shift to the affected side, a high diaphragm and varying degrees of collapse.

While the prompt use of suction to aspirate the fluid may avoid atelectasis, it is not sufficient to relieve a definite bronchial obstruction unless applied under endoscopic control.

Bronchoscopy should be instituted as promptly as possible. This should be done gently and rapidly so as not to subject the infant to

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From The Thayer Hospital.

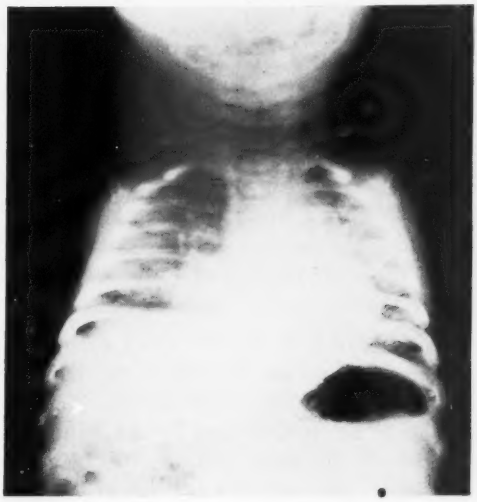

Fig. 1.—Roentgenogram showing atelectasis of left lung.

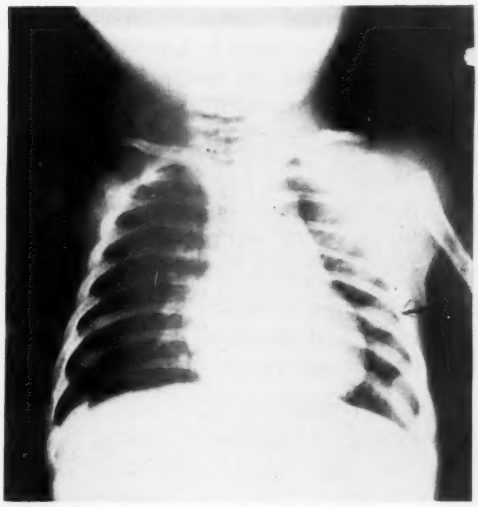

Fig. 2.—Roentgenogram after bronchoscopy showing clearing and return of the mediastinum to the midline and fracture of the seventh rib.

either trauma or a prolonged procedure. One minute should suffice to expose the larynx, insert a 3 mm. bronchoscope and free the plugged bronchus with a fine suction cannula. Once this is done the lung usually re-expands rather promptly. This can be verified by stethoscope and x-ray examination.

Trauma, incident to delivery, is not a common cause of respiratory complications. Some years ago I reported a fatal case of a ruptured trachea following a breech delivery done in a home, with resulting generalized emphysema. The following case of atelectasis is of interest due to the x-ray findings of a fractured rib which seemed to be the causative factor.

Baby William, age 4 months, was born prematurely at seven months, with apparently a normal delivery in another hospital. There were six other children in the family, all healthy. The child had difficult respirations since birth, was kept in the hospital, and was in and out of an incubator for three months. Oxygen was required a good deal of the time. The patient was referred to the Thayer Hospital for bronchoscopy. An x-ray film of the chest showed a marked atelectasis of the entire left lung with mediastinal shift to the left. In addition a healed fracture of the seventh rib on the left was noted.

Bronchoscopy revealed the trachea deviated to the left and the left bronchus somewhat compressed and obstructed by rather thick secretion. Obstruction was relieved by aspiration with prompt clearing of the lung and return of the mediastinum to its normal position. The baby made an uneventful recovery.

It must be presumed that the rib fracture, previously unrecognized, resulted from delivery and caused the atelectasis which persisted until bronchoscopic aspiration was done. It was fortunate that response to this procedure was effective after this four months' interval. Earlier attention would have been advisable.

All cases of respiratory difficulty in the newborn should be investigated with the idea of atelectasis in mind. Prompt bronchoscopic therapy will decrease both mortality and morbidity.

PROFESSIONAL BUILDING.

XXI

A CASE OF RHINOSPORIDIOSIS COEXISTING WITH LEPROSY

ERICH ROSENBAUM, M.D.

RAMAT-GAN, PALESTINE

An Indian soldier in his early twenties, G. S., reported sick on November 19, 1945, complaining of headache which had started about ten months previously. Approximately one month later a growth had developed at the entrance of his left nostril, the headache had increased and spread to the forehead, and with the onset of each painful attack his nose would be completely obstructed and he felt feverish. He stated he was an only son, his mother was alive and well, his father had died from plague.

On examination the patient's left nostril was totally obstructed by a polypus which almost protruded from the naris and could be easily seen by lifting the tip of the nose. This growth had the usual pale color of nasal polypi, but not the appearance of a simple polypus; its surface was not glossy and smooth, but more of a mulberry shape and looked rather granular at its posterior part when pushed laterally. It was tender to touch and bled easily. Contrary to the normal place of origin this singular polypus obviously did not arise from the ethmoid region, was not pedunculated, but sessile and arose in the anterior part of the left nasal cavity. It could be elevated laterally from the cartilaginous segment of the septum; hence it was improbable that it was a bleeding septum polypus. As I had never before seen such an extraordinary polypus and as the soldier was an Indian, I assumed it to be a tropical disease and had the diagnosis of nasal leprosy in mind. Therefore, I took a specimen from the left nasal fossa and since the nasal breathing was so much impaired, I booked the patient for removal of the growth at my next visit to the hospital.

The laboratory report of November 23, 1945, stated: "Acid-fast bacilli seen, resembling Bacillus leprae." Prior to the operation another specimen was taken.

On November 23, 1945, the operation was done under local anesthesia of cocaine-adrenalin. The tumor was fixed medially to the upper part of the septal cartilage, turned round to the vestibule,

where it was adherent to its upper wall near the mucocutaneous juncture, spread then towards the bottom to the left lower turbinate as well as to the mucous membrane and the inner skin integument of the ala nasi. The growth had to be partially loosened with a scalpel before it could be removed easily with a wire snare. There was no profuse bleeding and after removal the entire cavity could be inspected thoroughly. Nothing else abnormal was found, the middle meatus particularly was perfectly free. A small vaseline plug was inserted and the polypus sent for biopsy. The plug was removed the following day. The wound was healing well and good air passage was noted on November 26, on which day the medical-specialist reported: "Multiple patches of atrophic nature and anaesthetic to pain found on the back and top of the left shoulder; both ulnar and peroneal nerves show slight thickening. Diagnosis: Macular anaesthetic leprosy. Skin-sections of healthy and anaesthetic tissue sent for biopsy."

The laboratory report on the nasal tumor was: "A good example of rhinosporidiosis. Active sporangia are irregularly distributed in granuloma in various stages of evolution; there is no true evidence of neoplasia. Second nasal specimen: Scanty acid-fast bacilli seen. The Kahn test is negative."

A third specimen was taken. The laboratory report of December 2, 1945, noted: "Scanty acid-fast bacilli still present resembling Bacillus leprae; the histologic structure of the tissue of the skin section is that of a subcutaneous fibrosis with irregular thinning of the squamous layer; in the new connective tissue are foci of chronic inflammatory cells mainly grouped around small vascular lumina." This histology, though not specific, is said to be characteristic of skin leprosy.

The nasal cavity of the patient looked at this time quite normal; no nasal discharge was present at any time while he was under observation. The patient was transferred to the Skin Department and I lost sight of him.

DISCUSSION

After Seeber's first case report of rhinosporidiosis in 1896 Sharp and Biggs,[1] in 1944, estimated a number of less than one hundred known cases, whilst Meyer[2] and Habibi[3] counted 165 cases from the world literature until 1943-1944. Rhinosporidiosis is a tropical or subtropical disease caused by a fungal parasite of the class of the phycomycetes, occurring mostly in males and colored people; 16 of the cases mentioned in Habibi's report were encountered in Iran

alone. Arnold's and Whildin's[4] record is to my knowledge the unique one in which a white person (a boy aged eight years) was afflicted. No other coexisting diseases have so far been recorded, certainly no coexisting leprosy.

A classical description of rhinosporidiosis was given in 1928 by Benjamins.[5] As a conspicuous symptom of the disease, a granuloma appears in the nasal vestibule, usually on one side only. However, the lids and the conjunctivae were affected in some cases. Strong[6] quoted as an exception a case of Dhayagude's in a Hindu, in which the parasite was found in nodules scattered over the body and there was no nasal or nasopharyngeal growth. According to Benjamins' description the nasal polypus arises from the "anterior and upper part of the septum" and Spencer[7] reported also that the attachment over the quadrangular cartilage is "rather high or it may be at the anterior end of the inferior turbinate and to the turbinate." These statements can be confirmed by the findings in my case. With regard to the places of attachment and the whole appearance of this polypus I would rather refrain from classifying rhinosporidiosis as a subdivision of the "bleeding septum-polypi" (as Meyer does), which are more or less regarded as hemangiomatous and cavernous granulomata, for a histologic section of a rhinosporidic granuloma cannot be mistaken for anything else. I can desist from going into particulars as the photomicrographs in Habibi's publication are conclusive.

Figure 1 shows the typical consequences of the parasitic invasion by yet unknown paths, that is, numerous cystic sporangia which contain many thousands of nucleated spores. These endospores are liberated by rupture of the chitinous wall of the cyst, settle down in the connective tissue, and the cycle is repeated.

But in addition to these already well-known observations Figs. 2 and 3 show complexes of cell accumulations which have not yet been described in rhinosporidiosis. There are many large cells within the connective tissue looking like empty vacuoles, some with, some without a nucleus. which may be of quite irregular shape and found generally at the cell periphery. In some places one sees flakes within the cells which resemble curdled milk. All these large cells appear unstained and are so numerous that they can be visualized in all parts of the sections and, therefore, should not be regarded as artefacts. On receipt of these specimens I at first overlooked these cells but concentrated my whole attention on the sporangia and their attributes. With them the diagnosis of rhinosporidiosis seemed so incontestable that I dropped, of course, all my pride of having made

Fig. 1.—Photomicrograph showing the typical histopathology of a rhinosporidic granuloma with cystic sporangia in different stages of development. (X 84).

the diagnosis of leprosy, although it was obvious that the patient had once contracted leprosy. As bacilli leprae are usually found in sections and were absent here, it appeared quite improbable that the nasal tumor had anything to do with leprosy. I have been of the opinion that leprosy and rhinosporidiosis have been only coincident in this case, and it must be remembered that the last specimen taken three days after removal of the growth was positive for Bacillus leprae. Nodules and granuloma may be seen in leprosy but, to my knowledge, vacuolized cells have not been described in that disease.

However, a change of tissue structure into the described mucinous substance with these hydropic, foam-like, big, lucid and inflated cells has been described in another nasal disease, rhinoscleroma. These stain-resisting, swollen and dropsical Mikulicz cells have until recently, been considered specific for rhinoscleroma. Hanstedt,[8] in 1946, opposed this view and maintained that Mikulicz cells were not specific for rhinoscleroma, but were also found in other pathologic

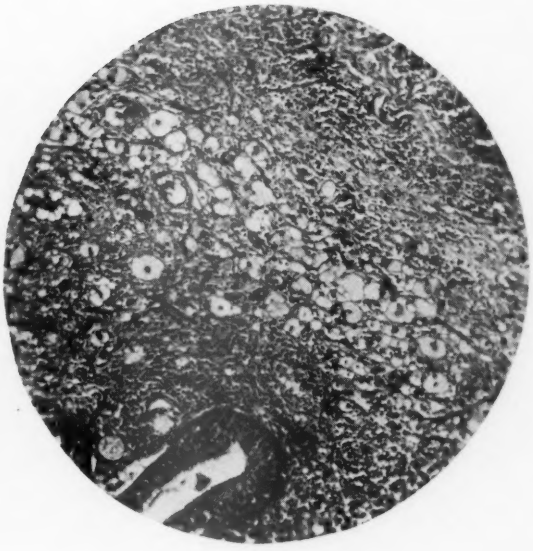

Fig. 2.—Photomicrograph showing the vacuolized cytoplasm within the connective tissue of a rhinosporidic granulomatous polypus. (X 84).

conditions. No report of reproduction of these cells in rhinosporidiosis has so far been published, but Whildin[4] mentioned that in Arnold's case there was a "foamy granular material" surrounded by the capsule of the sporangia and Spencer[7] first referred clearly to "vacuolated cytoplasm and a vesicular nucleus." If one's attention is focused to the vacuolated cells in Figs 2 and 3 and if one compares them with the Mikulicz cells in Falcao's photomicrographs[9] of a case of rhinoscleroma, the resemblance is so striking that Hanstedt's thesis is gaining support.

Not yet known is the mode of transmission of either rhinoscleroma or rhinosporidiosis. Almost all the lesions in rhinosporidiosis have been observed near the skin and in Dhayagude's case only at the skin proper. Scleroma develops also in the anterior portion of the septum or on the surface of the nose in the form of more or less subepithelial infiltration. As similar complexes of the described mucinous cells can be observed in either of these diseases,

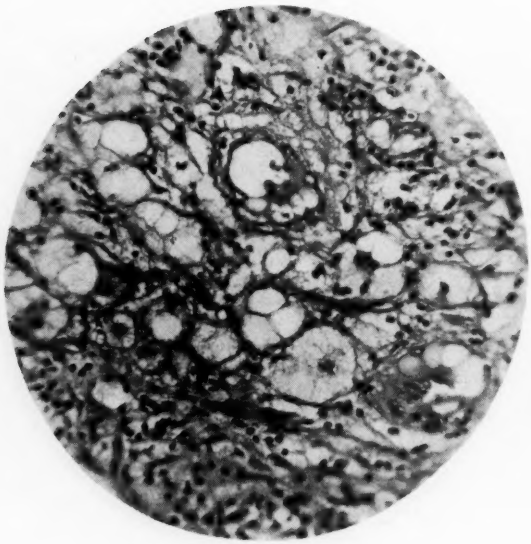

Fig. 3.—Higher magnification of the vacuolized cells. The majority of them show empty lumina, but filiform meshwork and curdled protoplasm can be seen in a few of them. (X 270).

the suggestion of a similar mode of infection presents itself. Should the Mikulicz cells actually not be pathognomonic of scleroma, but be found in various other infectious diseases of the nose, the toxic agent may be the product of some familiar infection which changes the mucous membranes into the mucinous cell groups, which in their turn may be the causative factors of a group of diseases. In that case these cells should be regarded as the cause rather than as the effect of such diseases. As this assumption is merely theoretical, a special search for Mikulicz cells should be made in diseases of thus far unknown origin.

Concluding his paper Falcao offered the suggestion that scleroma may be a general disease transmitted by mosquitoes and that the rhinolaryngotracheal lesions render the secondary stage of the disease very similar to leishmaniasis. The same could be said for rhinosporidiosis. I, therefore, conclude with Falcao's final words, that "col-

leagues, particularly those in the countries in which the disease is endemic, are urged to make further investigations in order to prove this concept!"

SUMMARY AND CONCLUSIONS

Though the rarity of cases of rhinosporidiosis alone justifies every additional contribution, the case reported here deserves special attention on account of a coexisting atrophic macular leprosy with positive nasal smears for Bacillus leprae.

Large vacuolated cells were found within the connective tissue which resemble the stain-resisting cells in rhinoscleroma. These cells have not yet been reproduced in cases of rhinosporidiosis. A yet unsatisfactory attempt has been made to explain the relationship of the probably identical cells in two different nasal diseases.

My thanks are due to my former assistant Captain S. K. Sen Gupta, IAMC, for invaluable help in translating and obtaining data, to the pathologist Major Gulerkary, IAMC, who furnished the sections and to Dr. Spanjer-Herford, Tel-Aviv, through whose courtesy the photomicrographs are reproduced.

56 HERZLSTREET.

REFERENCES

1. Sharp, H. S. and Biggs, R.: A Case of Rhinosporidiosis. J. Laryng. & Otol. 59:457-58 (Dec.) 1944.

2. Meyer, Max: Falle von blutendem Septumpolypen, verursacht durch Rhinosporidium Seeberi, Practica Oto-Rhino-Laryng. 5:139-43, 1943.

3. Habibi, M.: Contribution à l'etude de rhinosporidium Seeberi; Description de 16 cas observés en Iran, Practica Oto-Rhino-Laryng. 6:35-44, 1944.

4. Arnold, R. and Whildin, J.: Rhinosporidiosis of the Conjunctiva, Amer. J. Ophth. 25:1227-30 (Oct.) 1942.

5. Benjamins, C. E.: Die Tropenkrankheiten d. Luftwege und d. Mundhöhle, Hdb. d. Hals-Nasen-Ohrenh, Denker-Kahler, Berlin-München, Springer-Bergmann, 4:654, 1928.

6. Strong, R. S.: Rhinosporidiosis. In Stitt: Diagnosis, Prevention and Treatment of Tropical Diseases, 7th Ed., Philadelphia, The Blakiston Company, 1945, vol. 2, pp. 1154-1156.

7. Spencer, F. R.: Rhinosporidiosis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:580-82 (Sept.) 1946.

8. Hanstedt, Sten: Scleroma of the Nose and the Larynx, Acta oto-laryng. 34:339 (Aug.-Sept.) 1946.

9. Falcao, P. C.: Rhinoscleroma in Brazil, Arch. Otolaryng. 45:467-476 (April) 1947.

XXII

ANGIO-EPITHELIOMA OF THE POSTNASAL SPACE

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Phoenix, Ariz.

The patient was seen by his pediatrician of Prescott for a cold about the first week in May, and on May 24, 1944, the child began to complain of difficulty in breathing. On May 28, 1944, he was taken to the pediatrician's office, quite blue and dyspneic. It was at this time that a swelling was found in the right side of the throat. An attempt was made to open what seemed to be a peritonsillar abscess but no pus was found at this time or on several other attempts to open this swelling.

At this time sulfamerazine and sulfathiazole were used but no change was reported. On June 8, 1944, penicillin was started and he had 400,000 units given intramuscularly.

The patient was seen by an otolaryngologist in Prescott, who diagnosed the mass as a tumor and not an abscess.

The patient was sent to Phoenix, entered Good Samaritan Hospital on June 14, 1944, where I was called in consultation.

The physical examination showed an acutely ill, dyspneic, moderately well-nourished boy, four years old, who breathed with a snoring sound and suprasternal and supraclavicular retractions part of the time. There was no swelling about the neck. There was only a slight enlargement of the right anterior cervical glands. The hearing was somewhat impaired.

The eyes seemed to be normal in appearance and mobility. The fundi were negative.

Examination of the nose showed some stuffiness with mucopurulent discharge on the right side. Packs were used in each nostril for shrinking but it was found difficult to examine the back part of the nose except with a nasopharyngoscope, which revealed a nodular tumor on the right, almost filling up the right side of the postnasal space.

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Examination of the nasopharynx showed a bulging forward of the right side of the soft palate. The right anterior pillar had been perforated previously, in Prescott, when searching for a peritonsillar abscess. Some bloody discharge was coming from this opening. With my finger, I was able to feel this hard, nodular mass in the right pharynx, and without difficulty the tumor, which was loosened by previous procedures, was removed with some slight bleeding for a biopsy.

Examinations of the hypopharynx showed no abnormal growths or inflammatory changes.

The heart was somewhat rapid but there were no murmurs. The respiration was 16 per minute; no râles were heard. The nasal culture was negative for micrococcus catarrhalis. The red blood count was 4,350,000; hemoglobin, 91%; the white blood count was 11,200; polymorphonuclears, 89%; lymphocytes, 19%; eosinophiles, 2%. The urine was negative. The boy's temperature ran as high as 102° F.; pulse ranged from 100 to 140; respiration ranged from 10 to 28.

The pathological report was as follows:

Gross: The specimen consists of three masses of light pinkish-yellowish-gray tissue The tissue presents a nodular, somewhat hemorrhagic appearance. The appearance is that of a highly cellular and edematous tissue.

Microscopic: The section reveals endothelial cells showing a striking relationship to vascular spaces and channels. There is a marked preponderance of cells over vessels. Small arteries surrounded by several layers of large polyhedral fusiform cells showing degeneration and necrosis are seen. In some areas the tumor cells grow in diffuse sheets and in an irregular course resembling sarcoma. In these areas a pink-staining, intercullular substance is found. Hyperchromatism and mitotic figures are also noted. Other areas show an acute cellular infiltration with degeneration and necrosis.

Diagnosis: Angio-endothelioma (perithelioma), probably arising from the buccal mucosa of the soft palate.

The patient was given eight x-ray treatments at the Good Samaritan Hospital and at the Pathological Laboratory and told to return in one month.

The roentgenogram of the chest showed no metastases or bronchopneumonia at the time of examination on June 16, 1944.

The patient was returned to his pediatrician in Prescott, and it was reported to me on July 17, 1944, that he ate well, slept well,

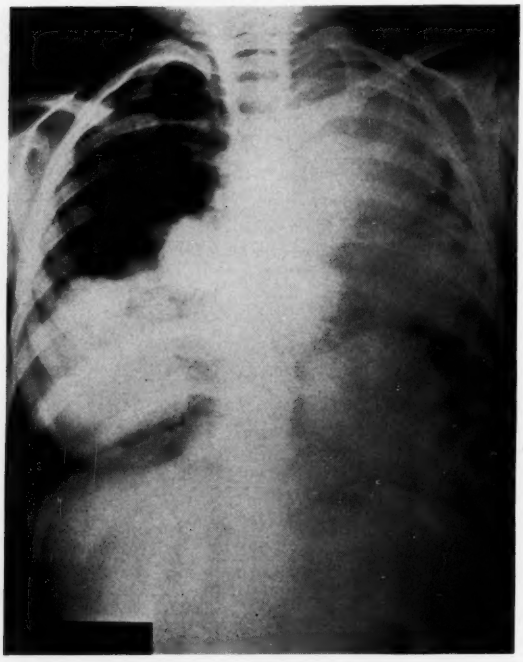

Fig. 1.—Roentgenogram showing metastasis of tumor in the lung.

breathed comfortably and seemed normal in every way and that he was to report to Phoenix on July 24 for more x-ray therapy.

The patient was seen November 16, 1944, by the radiologist, at which time another chest picture was taken at the Pathological Laboratory. At this time, the radiologist reported large areas of metastasis which were growing rapidly. He offered no encouragement about further treatment, as the densities developing in the lung certainly made the prognosis poor in this very interesting case.

There was further metastasis in the abdomen and the patient died the latter part of November 1944.

Comment. The tumor found in the nasopharynx of this four-year-old male child has been classified by the Army Medical Museum as an angio-endothelioma. They stated, however, that if that term is used to designate a malignant tumor, as it is in this case, they would prefer the diagnosis of angiosarcoma.

These tumors are composed of blood vessels in the early stage of formation.

Maurer,[1] in 1879, described two cases of slowly growing tumors composed of ingrowths of endothelial linings of blood vessels which he called angiosarcomas.

Angio-endothelioma of the nasopharynx is fairly rare. Salinger and Pearlman,[2] in a study of 24 cases of nasopharyngeal tumors, reported only one case of angio-endothelioma. Most of these cases do not produce symptoms referable to the nose and throat until quite late, so it is important that a thorough examination of the nasopharynx be made routinely in any case in which there is the slightest nasal obstruction or pains about the head and neck. If it is not possible to use a postnasal mirror then a nasopharyngoscope should be used. The important thing is to visualize thoroughly the postnasal space. This space has been called the "forgotten space." The pathology frequently found in this area certainly requires more attention than we usually give it in a routine examination.

Due to the anatomic situation of these lesions, they are difficult to treat. However, until the past year or two, the most satisfactory response was obtained from irradiation in the form of radium packs, applied externally, about the neck and head and placing radium tubes on a pliable applicator into the nasopharyngeal growth. During the past year, however, Figi[3] has been treating a group of patients with fractional dosages of roentgen therapy, according to the method of Coutard.

CONCLUSIONS

1. The postnasal space in the adult and the child should undergo more thorough and frequent examination for early detection of tumors, as earlier treatment means better prognosis.

2. Although angio-endotheliomas are rare and do not metastasize frequently, the majority become malignant (72 per cent of cases) and may metastasize as in this case.

3. Early treatment with x-ray and radium offers the patient his best chance.

4. The present status of nomenclature of postnasal tumors is unsatisfactory, as shown by wide disagreement among pathologists reviewing tumor slides and as brought out by Salinger and Pearlman.[2]

1011 PROFESSIONAL BUILDING.

REFERENCES

1. Maurer, F.: Ein Beitrag zur Kenntnis der Angiosarcome, Virchows Arch. f. path. Anat. 77:346, 1879.

2. Salinger, S., and Pearlman, S. T.: Malignant Tumors of the Epipharynx, Arch. Otolaryng. 23:149 (Feb.) 1936.

3. Figi, F. A.: Fibromas of the Nasopharynx, J. A. M. A. 115:665-671 (Aug. 31) 1940; Surg. Clin. North America, Aug. 1941.

XXIII

ANGIOSARCOMA OF THE PETROUS PORTION OF THE TEMPORAL BONE

REPORT OF A CASE

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TUCSON, ARIZ.

A white boy, eight years of age, was first seen by the referring pediatrician on May 14, 1943. The patient complained of severe pain in the region of the right mastoid, extending upward over the entire temporal area. The temperature was 102° F., the pulse rate 104. Palliative treatment was administered.

He was referred to the writer on the following day, and examination showed the patient to be a well-nourished boy weighing 66 pounds. The findings were moderate nasal and postnasal discharge and slight reddening of the nose and throat; the faucial tonsils were enlarged and infected; the right membrana tympani was thick, dull, and slightly injected, while the left was normal in every respect. Over the right mastoid bone there appeared a decided swelling, like a small bump. The boy explained that he had been struck two days previously with a rock and thrown down by another child. Two weeks previously he had suffered for two days from otalgia, right, but gave no history of an acute upper respiratory infection. Examination led to the following diagnosis: subacute inflammation of the paranasal sinuses; subacute pharyngitis and inflammation of the faucial tonsils; acute otitis media with mastoiditis, right; subacute cervical adenitis.

As the patient had a temperature of 101.4° F. and swelling and tenderness were noted over the mastoid and as the ear drum was definitely red and thickened, myringotomy was performed under ethyl chloride anesthesia. Sulfadiazine was prescribed, as well as sedatives and an ice bag was applied to the mastoid. The patient was ordered to bed.

The swelling over the right mastoid region decreased noticeably and the pain was alleviated. Smears made from the discharge of the

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middle ear after myringotomy revealed no organisms. The blood picture was: hemoglobin, 90 per cent; red blood count, 5,000,000; white blood count, 28,000 (polymorphonuclear leukocytes, 95 per cent; lymphocytes, 5 per cent). X-ray pictures of the mastoids showed the left mastoid to be normal; the right mastoid appeared rather densely sclerotic in its antral portion. Extension of density from the posterior superior apical cells suggested the possible presence of a peridural abscess. There was some indication of coalescence and breakdown in the region of the antrum. The roentgenological report stated that for a definite diagnosis further photographs would be required. However, such a course did not seem to be warranted as the patient made a fairly prompt recovery after myringotomy. The fever subsided on the second day; within nine days the ear canal was dry; 13 days later mastoid swelling and tenderness had completely subsided, the drum had healed and the patient was discharged.

The boy returned to his pediatrician five weeks later, on July 6, 1943, with a temperature of 98.6° F. Behind the right ear there was now encountered a pronounced swelling, which seemed to be fluctuating and to contain fluid. Upon incision by the pediatrician, a moderate amount of bloody serous liquid was released.

This serum was found to consist mainly of red blood cells, some lymphocytes, and only a few polymorphonuclear leukocytes. Neither bacteria nor acid-fast bacilli were encountered. Ten days later, the liquid had again collected in the cyst behind the right ear; after incision, about 5 cc. of blood-tinged serous fluid were removed. Yet, even after this intervention, the mass failed to subside to any noticeable degree.

Four weeks later the boy was again referred to the writer and presented at that time marked edema behind the right ear, which was not tender or red.

There was a large mass of granulation tissue protruding through the incision behind the right ear (Fig. 1).

A new set of x-ray pictures revealed a large oval defect, measuring 3.5 cm. in diameter, in the posterior portion of the mastoid bone and partly involving the occipital bone. At its peripheral margins, bone reaction and decalcification were clearly evident. The mastoid area itself appeared densely sclerotic. On the basis of these findings the following entities suggested themselves for differential diagnosis: (1) chronic mastoiditis combined with osteomyelitis; (2) Hand-Schüller-Christian syndrome; (3) tuberculosis involving the mastoid, with tuberculous osteomyelitis; (4) malignancy.

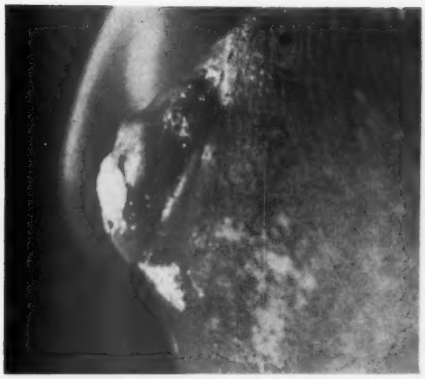

Fig. 1.—Large mass of granulation tissue protruding through the incision behind the right ear. (September 23, 1943).

During the following several days there occurred free discharge of serous fluid and the cyst decreased slightly in size. Specimens were removed from the granulation tissue for pathologic examination. Microscopic study revealed the presence of a malignant growth. The tissue specimens were quite cell-rich, the cells being of medium size, irregularly oval or triangular in shape. Occasionally, mitotic figures were encountered; there was a certain degree of amitotic division, but neither adenomatous structure nor true giant cells, lyphoid tissue or xanthoma cells occurred, and no resemblance to skin cancer could be established. On the basis of these findings the diagnosis of tuberculosis or granuloma was excluded, and the probable presence of sarcoma—possibly bone sarcoma—had to be assumed. The malignancy was of Grade 2 or 3, according to Broder's Index.

A complete blood count demonstrated a hemoglobin content of 75 per cent; red blood count, 3,900,000 (hypochromic); white blood count, 7,600 (polymorphonuclear leukocytes, 64 per cent; lymphocytes, 32 per cent; eosinophiles, 2 per cent; transitionals, 2 per cent). Wassermann, Kline, and tuberculin tests were negative.

On the basis of these reports, x-ray therapy was immediately instituted, and 3065 r. were administered by the radiologist to the site of the lesion in 21 treatments, extending over a period of 30 days. At each session, 150 r. in air were administered within six minutes, using 200 kilovolts and 10 milliamperes, through a regular cone and a filter equivalent to 1.65 mm. Cu., at a skin-target

Fig. 2.—The tumor mass, after a course of 21 x-ray treatments. (November 12, 1943).

distance of 50 cm. The size of the field was initially 10 cm., but later smaller fields of 8 cm. and 4.5 cm. were employed. The secondary infection responded favorably, beginning with the third dose of irradiation; after the seventeenth treatment the tumor mass was reduced to one third of its original size. Before termination of x-ray therapy, part of the remaining mass was removed by electrosurgery. However, an x-ray picture taken shortly after termination of irradiation treatment showed only little change in the appearance of the bone defect, as compared with earlier films.

In the course of the following months, the boy was regularly observed, and under local application of a sulfonamide to the wound, the discharge decreased considerably so that radiological treatments could be discontinued on November 12, 1943 (Fig. 2).

The boy returned to the writer for treatment on January 3 of the following year, with attacks of severe headaches on the right side and intermittent vomiting which had started four days previously. Edema was found in the right eye ground, but no diplopia was present. Nine days later, bloody serous fluid was discharged from the ear and paralysis of the right side of the face occurred. X-ray pictures taken at that time revealed a sharpening of the margins of the large oval bone defect but no further involvement of the bone by the neoplasm. Neurological examination disclosed complete paralysis of all muscles supplied peripherally by the right facial

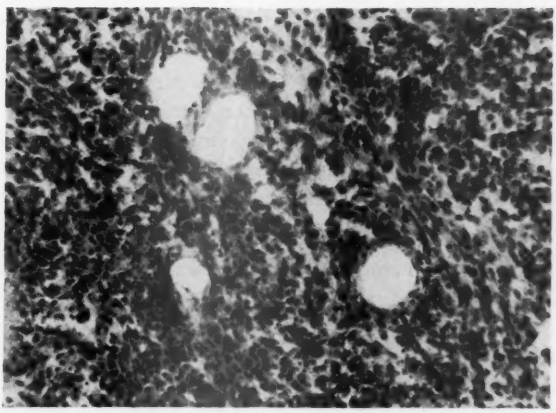

Fig. 3.—Photomicrograph of the tumor mass, diagnosed as angiosarcoma of the petrous portion of the temporal bone. (March 1, 1944). X 141

nerve, and impairment of the sense of taste in the anterior two-thirds of the right side of the tongue. There was noted definite elevation of the left optic disk, and hearing on the right side was markedly impaired. All other cranial nerves were functioning normally. On the basis of these findings, intracranial involvement originating from the lesion in the mastoid had to be considered, along with extension of the pathological process into the facial canal. A spinal tap revealed a pressure of 305 mm. of water; examination of the spinal fluid proved negative.

Radical mastoidectomy seemed inadvisable, as the prognosis in this case was very unfavorable, and a new course of palliative radiation was decided upon as a last resort. But before it could be started, discharge from the ear occurred, similar to that observed several months previously. The boy was ordered to bed and only general palliative treatment applied. On January 21, 1944, exophthalmos occurred bilaterally. The patient expired on February 29.

Autopsy, performed on March 1, demonstrated a tumor the size of a goose egg on the right side of the skull. Springing from the upper surface of the tentorium and from the bone in the posterior fossa of the skull close to the temporal bone, it evidently had pushed upward and formed a smooth cavity for itself within the brain. The

tumor was greyish-pink externally as well as on cut section; it was soft and showed no cystic areas. At its upper extremity it was slightly attached to the brain substance; at one point the tumor bulged against the cerebellum, yet there was no evidence of any connection with it. Through the floor of the skull in the mastoid region, there appeared an irregular opening, 2.5 x 1.5 cm., hemorrhagic in character, with smooth margins, and some bone spicules. No tumor was found either in the middle ear, in the ventricles of the brain, or in any abdominal organ.

A neurological examination of the tumor mass was performed. The external surface of the dura in the region of the mastoid had apparently extended by way of the middle and external ear and had formed a coat of tumor over the entire region, replacing to a considerable extent the mastoid cells. Within the intracranial space, the tumor had embedded itself in the temporal lobe, without invading the brain tissue proper. At its base, where the tumor involved the petrous bone, it measured approximately 2.5 cm. in diameter. A button of tumor, 1.5 cm. in diameter and 3 to 4 mm. in thickness, emerged from the porus acusticus internus and extended into the posterior cranial fossa.

Histopathological examination demonstrated the tumor to consist of many blood vessels or vascular spaces which apparently constituted an integral part of the growth. These spaces varied widely in size and were lined with modified endothelium. The intervascular tissue was moderately cellular, although in some areas it had been transformed into a connective tissue scar. At least in some portions of the tumor a fairly good connective tissue stroma was encountered. Some evidence of impaired circulation appeared in the formation of small areas of necrosis, some of which were suggestive of a pseudopallisading arrangement.

Under higher magnifications the tumor cells were found to vary considerably in size and shape. Many hyperchromatic nuclei and many typical and abortive forms of mitoses were seen. The tumor cells showed considerable variation in size and shape.

It must remain questionable whether this tumor arose from some portion of the arachnoid within the petrous bone (perhaps the porus acusticus internus) or from some structure native to the bone itself. The histopathological diagnosis was: angiosarcoma of the petrous portion of the temporal bone, possibly allied to the angiomatous group of meningiomas.

45 East Jackson Street.

XXIV

HEARING ACUITY VARYING WITH THE POSITION OF THE HEAD

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ST. LOUIS, MO.

The following case is reported less as a medical curiosity than to support the contention of Pohlman, Crowe, and others that impairment of hearing for high tones does not necessarily imply an inner ear or nerve lesion.

What concerns us about this patient at present is that he hears considerably better with his left ear while lying down than while sitting erect. The incidents in his past history which may have some bearing on the condition are as follows:

The patient, who is now 58 years of age, was first seen in 1931, when a persistent cough was eliminated by removing him from exposure to rabbit hair with which he came in contact as a salesman of felt hats.

In 1940 there was an edema of unknown origin about the orifice of the left eustachian tube, resulting in stuffiness which was quickly eliminated by the application of ephedrine.

In 1946, while in Chicago, he developed what was presumably a simple otitis media with fluid in the middle ear. Both drums were incised by a specialist in that city and when seen by me three weeks later, the left drum had not completely healed.

Late in 1947 after an airplane flight the patient again felt some stuffiness and reported that he could hear better while lying down. This statement was not seriously regarded at the time and owing to the appearance of the eustachian orifice and the repeated attacks of stuffiness, local radium application was resorted to, using the 50 mg. applicator with a .03 mm. Monel metal screen advocated by Crowe. Two applications of 12 minutes each were made with an interval of 14 days between. An audiogram made at this time showed an advancing deafness in the middle and upper range. As

he persisted in the statement that he could hear better when lying down, audiograms were made in the erect and supine positions with the results shown in Fig. 1.

A general examination made at this time was negative except for a periapical dental abscess. The tooth was extracted without change in the symptoms.

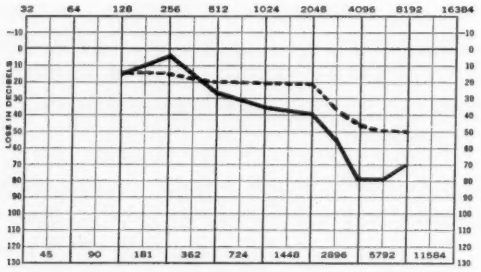

Fig. 1.—Audiogram, air conduction. Solid line, erect position; broken line, supine position.

The condition persisted in February 1948. At this time both drums were brilliant and thin. The only abnormality was that the incus could be plainly seen on the left through a thin area which remained after the paracentesis wound had healed. On inflation this thin portion ballooned from the remainder of the drum and there appeared to be some granulations on its tympanic side. While it was being observed the patient swallowed and the bulge returned to its retracted position. None of this had any effect on the hearing.

At times it was thought that a fluid level could be made out but this was never certain. Neither the bone conduction on the left nor any of the hearing on the right underwent any alteration with change of position.

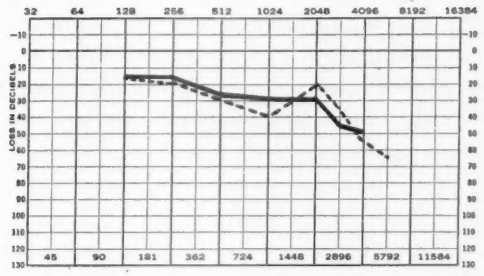

Fig. 2.—Audiogram, bone conduction. Solid line, erect position; broken line, supine position.

Comment.—Just what it may have been that altered the hearing on change of position is not too important. The significant thing to my mind is that in any event the alteration occurred in the conductive mechanism and that the resulting curve was typical of what we have been taught to regard as characteristic of nerve deafness.

Experimentally, the author had an audiogram of his own ear recorded and then repeated after instilling three drops of glycerine into the canal. The tests were made on the left ear while lying on the right. The results accord in general with those of the patient in the erect position as well as those observed by Bunch after filling the canals with water, in that the hearing at 256 d.v. and below remains approximately normal and that above that range the curve descends to a loss of 50 db. at 4096.

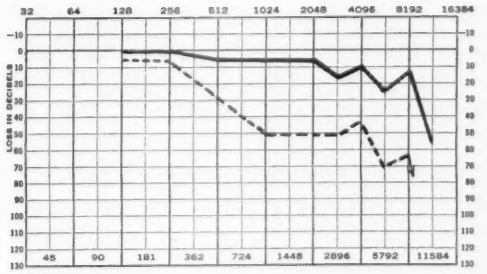

Fig. 3.—Experimental audiogram, author's left ear, air conduction, subject lying on right side. Solid line, canal unobstructed; broken line, after introducing three drops of glycerine into the canal.

If the acoustical results of fluid on the outer side of the drum are comparable with analagous conditions in the middle ear (and the present case would suggest that they are), then the differential diagnosis must depend upon the bone conduction tested with a tuning fork, rather than upon the audiometric curve.

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REFERENCES

1. Pohlman, A. G.: The Interpretation of Conduction Deafness, Arch. Otolaryng. 14:48, 1931.

2. Crowe, S. J., and Baylor, J. W.: The Prevention of Deafness, J. A. M. A. 112:585, 1939.

3. Bunch, C. C.: Clinical Audiometry, St. Louis, The C. V. Mosby Co., 1943, p. 74.

XXV

DIRECT LARYNGOSCOPY—A SIMPLIFIED TECHNIQUE. AN AID TO THE EARLY DETECTION OF LARYNGEAL CANCER

SAM ROBERTS, M.D.

AND

FRANK S. FORMAN, M.D.

KANSAS CITY, MO.

We are presenting these new instruments and improved technique for the following reasons:

1. Direct laryngoscopy with adequate exposure of the anterior commissure has been a difficult problem at times even for those surgeons in the larger clinics who have expertly trained assistants. It is always an enormous problem for those who work in smaller clinics, or alone, with untrained assistants.

2. We have been able to make the procedure so easy that every otolaryngologist will be able adequately, quickly and skillfully to expose all parts of the larynx where cancer or other tumors might exist. (It was our inability to do this that started the search for new and better methods four years ago.)

3. Direct laryngoscopy with older instruments has been so difficult that few otolaryngologists have ever perfected a satisfactory technique. They have depended on indirect laryngoscopy. With indirect laryngoscopy, cancer can only be "guessed at"; a specimen cannot be taken for biopsy.

4. Because of the difficulties, the art of direct laryngoscopy now reposes in the hands of a few laryngologists in the larger cities. Cancer of the larynx also occurs in the smaller communities where it must be diagnosed early if we are to reduce its tremendously high mortality rate.

It is impossible to teach the undergraduate student to make a satisfactory laryngeal examination. For many years we have, how-

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Presented before the American Academy of Ophthalmology and Otolaryngology, Chicago, Ill., October, 1947, under "New Instruments and Methods."

ever, been writing and rewriting on the blackboard the following statements for undergraduates:

> "A patient must never be allowed to be hoarse more than six weeks without a careful laryngeal examination."
>
> "It must be taken for granted that a patient who has been hoarse for six weeks has a cancer of the larynx, until it is proved otherwise."

If the general physician will remember these facts, and give this information freely to the general public, we believe that is all that can be expected of him. The disastrous delay in correctly diagnosing cancer of the larynx is the fault of the laryngologists.

There are three reasons why the laryngologist has failed to do good direct laryngoscopy:

1. Improper or insufficient anesthesia.

2. Lack of trained assistants to support the head in the correct position.

3. Lack of improved instruments.

The tests for a proper anesthetic in laryngoscopy are:

1. Complete abolition of the sphincteric action of the aryepiglottic folds.

2. Complete relaxation of all the heavy neck muscles.

It is a pitiful sight to see a laryngologist make one futile attempt after another to expose the anterior commissure of a nervous, struggling, gagging patient. In the end, both he and the patient are exhausted, the patient traumatized, and nothing accomplished. A few years ago we started simplifying the procedure.

Anesthesia is an important phase of the simplification. We have all been schooled too much in using only local anesthesia. Anesthesia, for any surgical procedure, must meet the following requirements:

1. The patient must experience no pain.

2. The patient must be completely relaxed.

3. The anesthetic must be safe.

With the majority of patients, the first two cannot be accomplished with local anesthesia alone. All patients, except those with respiratory obstruction, cardiac conditions or other contraindication,

should have both a local and a general anesthetic. Since the advent of curare and pentothal sodium we seldom use anything else for all our endoscopic work on adult patients.

Our anesthesia routine is as follows:

CHILDREN

A barbiturate is given according to age, weight, etc., one hour before operation.

The throat is sprayed with 2% pontocaine just before operation.

Open ether is used.

ADULTS

Delvinal sodium or other barbiturate is given by mouth ..... 1½ gr.

Morphine ..... 1/8 to 1/6 gr.
and
Atropine ..... 1/150 gr.
are given by hypodermic injection intramuscularly.

The above are administered one hour and 15 minutes before operation.

Twenty minutes before operation the throat is sprayed three times at five-minute intervals with 2% pontocaine, with the tongue held forward.

Just before intravenous anesthesia is started 5 cc. of 2% pontocaine are dropped into the larynx slowly, drop by drop.

The patient is taken into the operating room on schedule. The procedure is as follows:

Curare is given slowly in the vein, 60 to 80 units for the average sized patient. When the patient is unable to raise his head but a short distance from the table, ample curare for perfect relaxation has been given. Definite voice changes will also be noted.

At least five minutes should be allowed to elapse after curare is started before pentothal is given. *Never* give pentothal first.

Pentothal sodium 2.5% is given intravenously, very slowly, until the lid reflex is abolished. This usually takes another five

minutes. It is best to allow the anesthetist ten minutes or more.

Oxygen is given continuously through a tube in the side of the mouth.

The surgeon must await the signal from the anesthetist before the laryngoscope is introduced. If resistance is noted or if there is a sphincteric spasm of the aryepiglottic folds, more pontocaine must be sprayed on the cords from an atomizer, the scope must be removed and more anesthetic used, both local and general. When ample curare and local anesthetic are used, the dose of pentothal sodium is reduced by about one half.

With mutual understanding between the surgeon and the anesthetist as outlined above, the problem of anesthesia is solved.

The argument has been advanced that there are only a few anesthetists qualified to give curare and pentothal. We do not believe this is true. We work in four different hospitals using the "available" anesthetist. We have used this technique over 200 times without any serious difficulties, and at times when the patient was not considered a good surgical risk. It is true there is danger when any anesthetic is given and we must have trained anesthetists even with open ether.

Pentothal sodium 2.5% must be given slowly and with great care.

Prostigmine 1 cc. is given intravenously at the end of the operation to overcome the effects of curare. The patient is usually awake and able to converse in about 30 minutes.

To solve the second problem, proper head position, we devised mechanical head supports, modeled after the classical positions described originally by Chevalier Jackson.[1] Fig. 1 shows the head supports with an elevation of 15 cm. for adults, 8 cm. for adolescents and 5 cm. for infants; the angles are as shown by Jackson.[1]

We believe these supports are indispensable for those laryngologists who do not always have trained assistants available, for the following reasons:

1. These supports eliminate the most important man in the laryngoscopic team—the one who supports the head.

2. Trained helpers who really know how to support the head are seldom found outside the large hospitals.

Fig. 1.—Mechanical laryngeal head supports with sponge rubber.

3. The position being correct, only a most delicate lift is required to expose the larynx.

4. There is no stretching or bruising of the pharynx or the larynx.

5. The surgeon may press downward on the patient's forehead and elevate the larynx more completely.

6. There is at least 5 cm. lateral movement inside the support, sufficient to expose ventricular lesions.

7. The possibility of trauma to the neck is practically eliminated, because no rigid structure comes into contact with the patient. Even though the canvas is heavy, there is ample "give" under the neck, and when covered with sponge rubber it is not uncomfortable.

8. Elevation and angles are always correct, if the right sized support is used.

9. The supports eliminate the human element of inattention, or inefficiency. This is important at critical times in removing small or inaccessible tumors.

10. The support may be removed at any desired time and manual support substituted, although we have never found this necessary for laryngeal work.

11. The supports were designed for laryngeal work only, but we also use them to expose the larynx before introducing the Jackson bronchoscope.

We frequently use the adolescent size head support for small adults.

Fig. 2.—The Seiffert laryngoscope.

We find the three sizes sufficient and have never had to change sizes after starting the operation.

To attempt to solve the third and last problem—namely, lack of improved instruments—we have devised a self-retaining dual distal lighted laryngoscope and designed a vocal cord spatula.

So far as we know, Seiffert was the first to design a self-retaining laryngoscope making use of the chest arm (Fig. 2). He had no lighting system. Haslinger later improved this by proximal lighting and a more adjustable chest arm.

THE SELF-RETAINING LARYNGOSCOPE

We have retained the chest arm of Seiffert and Haslinger. (Fig. 3).

After many consultations with lighting engineers, we have had installed in the new instrument dual distal lighting. We were told by these engineers, and have proven it to our own satisfaction, that any single light at the end of a tube throws a shadow. With lights on both sides of the distal end of the tube the interior of the larynx is perfectly and uniformly illuminated without shadows.

The next problem was reflections from inside the tube. These reflections, when actually measured, were enough to interfere materially with the surgeon's vision.

For the above reasons, we have had the inside of the tube of our laryngoscope processed in black. It is a great comfort to work without the distracting annoyance of shadows and reflections.

We have put lead in the handle to give the instrument a better balance. This extra weight also helps to maintain a more constant and even pressure inside the larynx above the anterior commissure.

We have opened up the sides of the tube for better intake of oxygen.

We have elevated the tip of the tube to give better visualization of the anterior commissure. This has been described by Holinger and others. With the tip elevated, the curvature of the distal end of the tube is so much decreased that we are now able to use the same tube for both adults and children as young as seven.

DIRECTIONS FOR USING THE SELF-RETAINING LARYNGOSCOPE

Anesthesia is given as described above.

The patient's head is placed on the laryngeal head support with the chin pointing directly upward (Fig. 4).

The self-retaining laryngoscope is introduced into the oral pharynx in the midline (or at the side of the mouth if the front teeth are prominent) the same as any other laryngoscope. As the epiglottis is engaged, the handle is raised, lowering the tip, and the whole instrument is pushed forward until the posterior commissure comes clearly into view. The chest arm (Fig. 3, *3*) is then released by the set screw (Fig. 3, *2*) and drops onto the patient's chest where it is locked by the same screw. The anterior commissure is now exposed by turning the thumb screw (Fig. 3, *4*), which raises the tip of the laryngoscope in the direction of the arrows. It will be noted that the pressure is exerted only on the anterior surface of the larynx and on the chest.

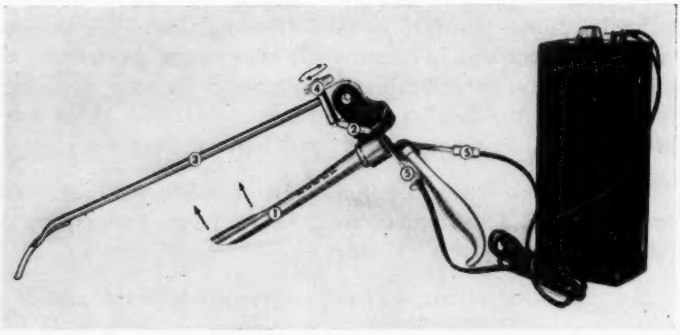


Fig. 3.—Self-retaining, dual distal lighted laryngoscope. *1*—Perforated tube to allow better oxygen intake and elevated tip to increase exposure of the anterior commissure. *2*—Set screw, for quick release and fixation of the chest arm. *3*—Chest arm, which fixes the tube in the larynx, making it self-retaining. *4*—Thumb screw, for elevating the tip of the laryngoscope in direction shown by arrows. *5*—Dual distal lighting, from battery. This eliminates shadows by illuminating the lesion from two sides.

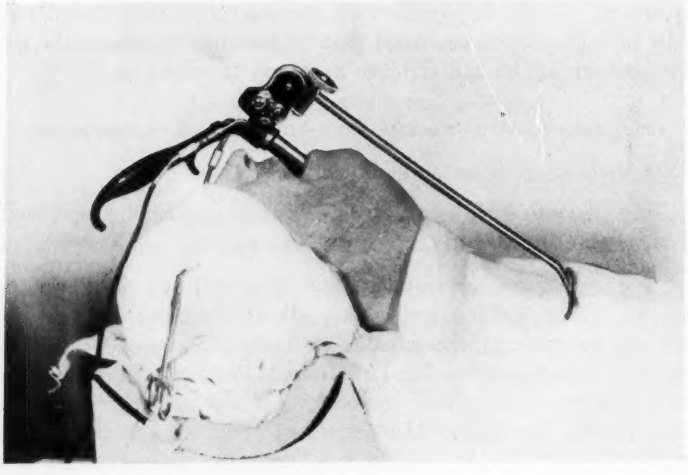


Fig. 4.—Self-retaining laryngoscope in position. The larynx is completely exposed. The chin must be pointing directly upward.

When the operation is completed, 1 cc. of prostigmine is given intravenously. For best results this entire technique must be followed. The head support and the self-retaining laryngoscope may be used with local anesthesia only, but we do not think it is as satisfactory.

We know this laryngoscope with mechanical head supports will greatly simplify the technique of direct laryngoscopy. It will also simplify the teaching of laryngology. Always before the student or the visitor would get only a glimpse of the larynx—over someone's shoulder. Now, the surgeon may place the laryngoscope in position, fix it with the self-retaining arm, step aside and allow the visitor as much time as necessary for complete study.

We know of no other technique where this would be possible, except perhaps suspension and this would require a constant changing of head mirrors.

We realize there are a number of experts doing suspension with splendid results. In our hands, however, there have been so many technical difficulties and so much trauma that we have abandoned its use altogether.

Vocal Cord Spatula.—We devised this spatula to overcome some of the jumping of the vocal cord, due to electrical nerve and muscle stimulation when diathermy is used. It is also finished in black to eliminate reflections (Fig. 5).

The instrument is placed between the cords with the cup under the cord with the lesion. It is sufficiently thick at the base to keep the opposite cord well away from the involved cord and the electrode. We experienced some difficulty at first in getting it made of nonconducting material and of sufficient rigidity not to bend into the direct line of vision. This difficulty has now been overcome.

SUMMARY

Direct laryngoscopy with *complete* visualization of the entire larynx has been a difficult procedure. Even those of great experience and with trained assistants have failed, at times, to get an adequate exposure of the anterior commissure.

Notwithstanding all the smiling faces one may see in the motion picture films after a laryngoscopy under local or no anesthesia, most patients say it is a very painful, disagreeable procedure. Children are just as sensitive to pain as adults. Some form of general anes-

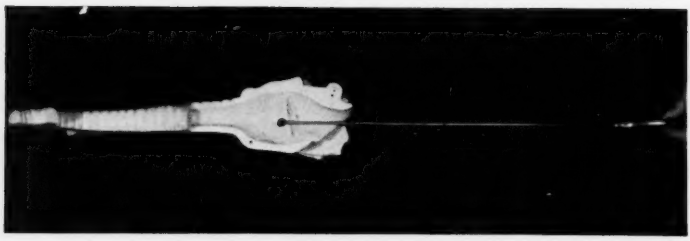


Fig. 5.—Vocal cord spatula.

thetic should be used in nearly all endoscopic procedures unless there is some definite contraindication.

Local anesthesia plus curare and pentothal sodium is ideal in most adult cases. It is also more humane. The anesthetist should be allowed ten minutes or more from the time he starts the curare before the laryngoscope is introduced. If muscular rigidity or laryngeal spasm is encountered, withdraw the laryngoscope, give more anesthetic, both local and general, and await complete relaxation before reintroducing the laryngoscope. It saves time and effort later.

Never be alarmed about the patient's condition when you encounter laryngeal spasm; it means too little, not too much anesthetic. Neither should you be alarmed at shallow breathing; this is to be expected and the reason for giving continuous oxygen.

You must work deliberately and insist on ideal conditions.

A hurried laryngoscopy is nearly always a bad laryngoscopy.

Trained assistants are seldom available except in large hospitals.

The mechanical head supports eliminate the necessity for the most important assistant.

The self-retaining laryngoscope with dual distal lighting eliminates shadows and increases the illumination.

Processing the inside of the tube of the instrument in black eliminates reflections.

The fixation arm makes it self-retaining.

These features incorporate so many advantages over other instruments that, in our opinion, it is indispensable to every laryngol-

ogist. It relieves the surgeon of an enormous physical strain, especially in his left arm. Several times we have heard the chief ask a younger and stronger assistant to support the laryngoscope because his left arm was exhausted. This does not make for good laryngoscopy because the assistant is doing his job blindly.

When the self-retaining laryngoscope is used, the surgeon can work with both hands free.

The battle is at least half won when any malignant growth can be located, either by x-ray or indirect visualization.

Never attempt any surgery on vocal cords until they are perfectly relaxed and stand widely apart. A biopsy specimen cannot be taken with any degree of accuracy when the cords are approximated or in a state of spasm. Many a patient with cancer has been lulled into a sense of security and later lost his life because of a negative biopsy of tissue taken from the wrong place.

This is well illustrated in "The Story of a Victorian Tragedy."[2]

" . . . triumph and disaster that made Morell Mackenzie's name remembered and was to end in tragedy for the prince and physician alike."

The great pathologist, Rudolf Virchow, a personal friend of the prince, would not have missed the diagnosis had he been given the proper tissue. One can well imagine that the whole history of Europe might have been different had Morell Mackenzie, the famous English laryngologist secured an accurate biopsy specimen. Crown Prince Frederick who had cancer of the larynx reigned, a sick man, only 99 days as Emperor Frederick III of Germany. He was a great liberal with a fine religious training. He might have ruled Germany successfully many years without his larynx. Many of our patients carry on a normal business and social life with a similar handicap. Frederick could have at least postponed for a while the iron rule of Kaiser Wilhelm II (First World War).

When the technique here presented is followed in every detail, the teaching of laryngology will be on a more scientific basis.

Finally, good laryngoscopy should not be in the exclusive hands of a few in the larger cities. Cancer also occurs in the smaller communities where it must be diagnosed early.

Every otolaryngologist should now be able to do a good direct laryngoscopy, to expose completely every part of the larynx, and to secure an accurate biopsy specimen.

This article is presented with that objective.

We wish to acknowledge with much appreciation the great help given us by Paul H. Lorhan, Associate Professor of Anesthesiology, University of Kansas School of Medicine.

1110 Professional Building.

REFERENCES

1. Jackson, C., and Jackson, C. L.: The Ear, Nose and Throat, Philadelphia, W. B. Saunders Co., 1945, p. 616.

2. Mackenzie, Morell: The Story of a Victorian Tragedy, London, Heinemann, 1946, p. 71.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, February 3, 1947

(Concluded)

THE PRESIDENT, DR. FRANCIS L. LEDERER, IN THE CHAIR

Lymphosarcoma of the Tonsil: Case Report

S. M. MORWITZ, M.D.

(This paper appeared in full on page 892 of the December 1947 issue.)

DISCUSSION

DR. ALFRED LEWY: I should like to report the case of a woman who was in her 50's and complained of deafness for which she had been treated by inflation, which gave temporary relief. She had a postnasal discharge, at times bloody. I found a tumor with superficial ulceration in the nasopharynx, biopsy of which showed lymphoepithelioma, and she was referred to the tumor clinic for irradiation. Involvement of the cervical glands was questionable; I thought I did palpate some glands but they were not hard and were freely movable; these glands were included in the irradiation therapy. Within six or eight months there was no tumor to be seen in the nasopharynx; there was some scarring covered by intact mucosa. However, as a result of irradiation she developed suppuration of the middle ear and the canal with more or less distortion of the auricle and some superficial lesions of the skin around the ear. This yielded to local treatment.

This woman lived for more than two years, during which interval she developed one large cervical gland, a biopsy of which was not done. It was treated by irradiation and reduced in size. She died of pneumonia. An x-ray of the lung taken one month before pneumonia developed was said to be negative for any appearance of malignancy in the lung. I did not see the x-ray film.

DR. FRANCIS L. LEDERER: Drs. Holinger and Rigby reported the case of a sarcoma of the larynx in an infant, but this is the first case that I have known of in the Clinic in one so young.

Meeting of Monday, November 3, 1947

THE PRESIDENT, DR. JOHN R. LINDSAY, IN THE CHAIR

The Use of Pyridoxin (B-6) In Vertigo: Clinical Note

ALFRED LEWY, M.D.

AND

NOAH FOX, M.D.

(Abstract)

No attempt is made to describe the pharmacology. Pyridoxin has been used empirically and with considerable success in such apparently unrelated conditions as granulocytopenia, vomiting of pregnancy, radiation sickness, Parkinson's syndrome.

Eighty-seven cases are reported; of these 17 have no final reports and are for statistical purposes classified as failures. Forty-seven are classified as Ménière's disease, that is, the lesions were peripheral and noninflammatory. Fifteen patients in this group had previously been unsuccessfully treated by means of salt prohibition, ammonium chloride, and dehydration, but responded to pyridoxin. Three cases were frank failures with the patients on pyridoxin. Several had recurrence of vertigo on stopping treatment, which disappeared on reinstatement of B-6. Some patients showed an erratic audiogram of the affected ear which leveled out after treatment. This supported the diagnosis of a peripheral lesion.

The presence of vertigo without evidence of either a peripheral or a central lesion is suggestive of a general cause, toxic, vasomotor or allergic. There were 40 such cases. No cases were positively diagnosed as organic intracranial lesions.

These were all private cases and could only be observed clinically. The authors realize that the causative factor should be diagnosed if possible, but in the absence of such diagnosis pyridoxin may be used empirically.

The dosage was 100 mg. per day in divided doses. For quick effect in severe cases the initial dose of 100 mg. was given intravenously.

DISCUSSION

DR. THOMAS GALLOWAY: What is the pharmacologic action of pyridoxin? Where specifically does it act?

DR. FRANCIS L. LEDERER: It seems unfortunate that in otolaryngology we must indulge ourselves in so much empiricism and carry out therapy for which there is no apparent logical basis. Pyridoxin employed in the treatment of vertigo is an agent for the action of which we seem to have no explanation. We have employed pyridoxin in the treatment of vertigo in a number of cases, and I feel that Dr. Lewy's presentation is very conservative. Certainly, in our own experience, we likewise have had some good results. Of course we must always remember that the definition of what constitutes true Ménière's syndrome leaves much to be desired. Many cases are referred with that diagnosis and every possible labyrinthine dysfunction may be involved. Unless we base our interpretation on better grounds, therefore, a good many patients will be treated in this empiric fashion and there may be difficult times ahead for us. All one can conclude at the moment is that pyridoxin, whatever its action, does have a beneficial effect in some cases of vertigo.

DR. GEORGE WOODRUFF: I have had no experience with pyridoxin in the treatment of vertigo, but I believe Dr. Lewy mentioned that these patients had been treated with salt-free diet and other therapy. Dr. Williams of the Mayo Clinic has said that instead of a salt-free diet and ammonium chloride he used fairly large doses of potassium nitrate, and when this was done, particularly in endolymphatic hydrops, it was unnecessary to limit the salt. I have found potassium nitrate of some value in certain cases.

DR. JOHN R. LINDSAY: It would seem that a remedy that would appear to have such wide application as this should be worth looking into. I would like to ask Dr. Lewy if he makes any distinction between vertigo from different causes, or do I understand correctly that treatment has proven effective in cases of central origin as well as of peripheral origin? I wonder if he has treated patients who have postural vertigo from multiple sclerosis or some other known neurologic disease; does it relieve the symptoms in such cases? It is a little difficult to appreciate any single drug affecting the basic pathology in true Ménière's disease which is a hydrops of the labyrinth. The vertigo in this condition is unpredictable, occur-

ring at irregular intervals which may vary from a matter of hours to many months.

Evaluation of therapy on the basis of attacks of vertigo alone in Ménière's disease is therefore not reliable. Auditory symptoms must also be considered.

DR. O. E. VAN ALYEA: I would like to ask Dr. Lewy how long he continues treatment in those cases which do not respond rapidly to pyridoxin therapy.

DR. SAMUEL SALINGER: I think there are two important items that need clarification in order to fully appreciate this report. First, what is the pharmacology of this drug and what is its action? Second, in what particular type of case is it indicated, and why? Is it possible that Dr. Lewy might give us some additional information on these points?

DR. ALFRED LEWY (closing): I anticipated most of these questions; I am unable to answer most of them. I know absolutely nothing about the pharmacology of pyridoxin and have been unable to find anything in the literature concerning its action in vertigo.

In many of the cases I was unable to find the cause of the vertigo; 47 were classified as Ménière's disease. We believed the lesion to be preipheral, presumably ectasia of the labyrinth. Of the general causes, we do not know whether it was due to direct toxemia or vascular disturbance acting on the vestibular mechanism. In none was there a diagnosis of a space-occupying lesion. It would be interesting to know if pyridoxin is effective on that basis.

In a symposium before the American Otological Society last April, Levy and O'Leary described eight cases of platybasia, meningioma of the cerebellar pontine angle, aneurysm of a circumferential artery, multiple sclerosis, vertiginous epilepsy, migraine with vertigo, hypothyroidism, vertigo and personality disorder, and vertigo followed by anxiety hysteria, respectively. I have had no opportunity to try this in vertigo from multiple sclerosis or epilepsy. I have had cases of migraine with vertigo; the vertigo was relieved but not the migraine. I have had no opportunity to try it on any cases of proved organic intracranial lesions. Dr. Furstenberg, who summed up the symposium, laid emphasis on the psychiatric element—for instance, in the case of anxiety hysteria and the personality disorder.

I am simply giving the clinical facts. In many cases that are seen we cannot make a diagnosis and the neurologists cannot make a diagnosis. Sometimes a focus of infection, such as gall bladder disease, is found, and treatment directed to that source often helps. We have cured many patients without making a diagnosis.

If I do not see improvement fairly promptly, I do not continue to administer pyridoxin. Some patients have improved almost immediately; others may not show improvement for several days. I certainly would not carry it on for three or four weeks if no improvement is apparent. Incidentally, it is quite expensive.

I do not know what pathologic conditions are favorably affected.

DR. FRANCIS LEDERER: Have the essayists had any personal experience on this subject?

DR. GEORGE WOODRUFF: Did any cases relapse following treatment?

DR. ALFRED LEWY: One of the essayists had a Ménière's attack that was classical, the attack went almost to unconsciousness, with absolute inability to move, with vomiting, nystagmus, complete prostration, and he was back in the office in three weeks.

Several patients showed a relapse on stopping treatment, but when it was resumed the vertigo disappeared. On the second occasion I have given the same dosage. Perhaps larger doses would help in some cases that were failures, and perhaps some would get along on lesser doses. I have seen no ill effects after a period of five or six weeks, but I read of one death attributed to anaphylaxis following a second intravenous injection of pyridoxin; I could not locate the original publication. I have noted no ill effects, not even side effects.

Except in one case, I cannot say that either the tinnitus or the deafness was markedly improved.

I believe the product is entirely synthetic. I could learn nothing on the pharmacology—everything I have seen was in chemical formulas.

Bone Conduction Observations in Fenestration Cases Physiologic Considerations

ARTHUR L. JUERS, M.D.

(Abstract)

(This paper appears in full on page 28.)

DISCUSSION

DR. PAUL CAMPBELL: The Weber test, as we all know, involves function of both ears, not just one ear. The result of this test is thus influenced by the situation in the unoperated ear. I would like to know how Dr. Juers controlled the unoperated ear in this study.

DR. ROBERT LEWY: It is a common observation, if one has two normal ears and does the Weber test with the finger in one normal ear, the Weber test will lateralize to the side where the "conduction deafness" is thus produced. This being the case I cannot understand why the packing placed in the fenestrated ear would not in itself produce an identical result.

DR. MAURICE SNITMAN: Did Dr. Juers state that slight pressure on the pars tensa produced vertigo by compression inward of the round window? From personal experience I have found such pressure on the drum produces a tug on the tympanomeatal flap with consequent pull on the underlying membranous canal with resultant vertigo.

DR. H. F. KOBRAK: This is an excellent paper which contains not only a comprehensive collection of previous work on bone conduction but also much new information and new interpretations. The remarks made on the Weber test corroborate what we have known in the past: the Weber test is unreliable and not valuable except in one condition, the shift from the bad ear to the good ear when a labyrinthitis develops. Dr. Juers has shown that this test holds for the fenestrated ear.

I was very glad to hear Dr. Juers' opinion on true increase of bone conduction in case of middle ear deafness. He mentioned the work of Herzog and Krainz. In my opinion their experimental work on models and on fresh temporal bones is the most elegant explanation of the increase of bone conduction.

In his last slide Dr. Juers showed a sketch of the middle ear in which the longitudinal air waves were drawn as parallel lines showing areas of compression and rarefactions, as they occur in a sound wave. It seemed to me that these lines indicated a too short wave length compared to the size of the ear. An air-wave of the frequency 1000 is about a foot long. This means that the important frequencies have a wave length which is large in comparison with the structures of the ear. All studies on phase relationship between oval window, round window and the new fenestra must be based on this physical fact. Maybe Dr. Juers could explain this slide a little more fully.

DR. GEORGE SHAMBAUGH, JR.: Dr. Juers has been working on this problem which is of considerable practical application as well as of theoretical interest. We have tried to find some practical method of detecting labyrinthitis on these patients who have had fenestration operations, and in a series of cases we made daily bone conduction audiometric tests in order to follow the cochlear function. We found the Weber test the most practical way to detect labyrinthitis. Second, the mechanism of hearing after fenestration is still as much of a mystery, but it is important. There are about five per cent of patients who are apparently ideal for operation but who fail to get any improvement. There is something about the physiology of hearing in these cases which is not yet understood but which should explain the failure. I think future progress in the fenestration operation will come largely from the study of the physiology of hearing after operation in order to permit us to get the maximum hearing improvement in our operations.

DR. ARTHUR L. JUERS (closing): In reply to Dr. Campbell's question, we have no way of knowing what is happening in the unoperated ear, but in trying to find a test of value for detection of early cochlear depression it is not unreasonable to assume that the unoperated ear is unchanged. The Weber test is not infallible by any means. Dr. Lewy's question concerned the packing's producing lateralization to the operated ear; that thought occurred to us. I tested routinely 50 patients coming through the office and in approximately two-thirds the test still lateralized to the operated ear. The majority had obtained serviceable hearing and were completely healed.

Dr. Snitman asked about the fistula response to pressure against the pars tensa. That response is opposite to what we get when pressure is applied over the fenestra. Movement is probably transmitted

through the tympanomeatal flap rather than through the round window.

In answer to Dr. Kobrak's question regarding the wave length represented on the slide—this was just a sketch to show the possible relationship of sound phase to the two windows and the usual proportion of wave length to actual cochlear dimensions was not considered.

Abstracts of Current Articles

EAR

Audiometric Studies of a Patient with An Artificial Ear Drum.

Aubry, M.: Les Annales D'Oto-Laryngologie 64:167-168 (March-April) 1947.

The author presents a very interesting set of observations in the case of a patient having a large perforation in the ear drum on the right side. There was a moderately severe conduction deafness in this ear, with lateralization of the Weber test to the right. The left ear had a much more severe degree of deafness and was not considered in the study. After the placement of an artificial ear drum over the perforation in the right tympanic membrane, there was found to be a significant gain in hearing in this ear: from 14 to 20 decibels through the conversational range, for air conduction, which brought the hearing up within useful levels. Perception of the whispered voice was improved from a distance of 25 centimeters up to more than 2 meters. Audiograms using a bone conduction receiver showed no significant change in the threshold for hearing after placement of the artificial ear drum.

HARKINS.

Surgery of Deafness.

Aubry, M.: Les Annales D'Oto-Laryngologie 64:5-25 (Jan.-Feb.) 1947.

The author traces the historical development of the fenestration operation and presents six cases of his own. In general, this worker follows the technique of Lempert, with the following variations. The skin and periosteum of the superior aspect of the external auditory canal are dissected free and a trephine opening made into the epitympanic space. The author stresses the point that he does not enter the mastoid antrum nor the aditus-ad-antrum. He removes the head of the malleus and the incus. He makes his fenestra in the ampulla of the external semicircular canal. He places the tympanomeatal flap over the newly made fenestra and the inner aspect of the attic in such a way as to close off the aditus, and thus excludes the mastoid cavity from the operative cavity.

Of the six cases presented, with audiometric findings, three patients obtained no significant gain in hearing. The other three did

obtain good results for the return of useful hearing in the operated ear. In one of these cases the author used a small Thiersch graft to cover the fenestra, which procedure was successful.

HARKINS.

The Peep-Show.

Dix, M. R., and Hallpike, C. S.: Brit. Med. Journal, Nov. 8, 1947.

The authors call attention to the difficulty of evaluating the degree of hearing impairment in children under six regardless of whether speech or pure tone audiometry is employed. Because of the fact that severe hearing impairment in these children is frequently accompanied by speech defects and because vocabulary is frequently limited, they have concentrated on pure tone audiometry. However, they state that pure tone audiometry, as practiced with adults, is impossible in these youngsters because of lack of cooperation. They have, therefore, devised an apparatus with which they can test the child with both visual and auditory stimuli at the same time.

Their apparatus consists of a viewing cabinet into which a child looks. At the top of the viewing panel is a loudspeaker. Just below this is a peephole which can be closed at will and behind which is an 8-volt lamp. Just below the aperture through which the child looks into the viewing box is a switch. An assistant sits beside the child.

The operator is behind a screen which has a peephole through which he can see the child without himself being seen by the child. He has two switches, one of which sends a selected pure tone into the loudspeaker at any desired intensity level, the other switch turning on the light before-mentioned. When he operates these two switches simultaneously, the light on the view box flashes and a pure tone sound comes from the loudspeaker. At this point the assistant beside the child presses a switch on the view box which lights a second 8-volt bulb in front of the picture of some familiar animal in the view box. She then signals the tester to cut off the stimuli. The tester repeats this maneuver a few times and soon the child no longer waits for the assistant to press down the switch on the view box which lights up the animal inside the box but as soon as the simultaneous visual and auditory stimuli are received, the child himself will reach for the switch in order to see the picture of the animal within the view box.

When this routine has been carefully established between the tester and the child, the nurse or assistant quietly closes the slot over the peephole in the view box behind which the light stimulus is located. The tester continues to send the pure tone through the loudspeaker, gradually reducing its intensity and if the child hears the tone, he knows it is time to press the switch which illuminates the animal picture in the box. By using a different animal picture for each of the eight tones of the audiometer, the child's interest is held and a graph of the intensity level at which the child hears each pure tone can be made.

The authors believe that pure tone audiometry accurately applied as by the peep-show procedure certainly yields a quantitative assessment of hearing which is considerably more accurate than anything which can be achieved by means of pitchpipes, drums or the spoken voice. The responses of the child can be inculcated by anyone able to secure the child's cooperation in simple play. The total time for the test procedure rarely exceeds fifteen minutes.

GROVE.

NOSE

Excessive Self-Medication with Naphazoline Hydrochloride (Privine Hydrochloride).

Mertens, P. S., Jr.: J. A. M. A. 134:1175 (Aug. 2) 1947.

Rhinitis medicamentosa is a condition frequently associated with allergy or sinusitis. The author has noted that this condition is rapidly increasing in medical practice due to excessive self-medication with "privine hydrochloride." This produces a severe rhinitis with a gray-colored mucosa of a most refractory nature with a more severe form of nervousness than one would expect to result from nasal obstruction alone. It is easily diagnosed by history and examination but must be distinguished from its complications. Treatment, which consists of withdrawal of the medication, will result in a cure.

BOIES.

PHARYNX

Tonsil Stumps.

Current Comment: J. A. M. A. 134:698-699 (June 21) 1947.

Tonsillectomy is the most frequent surgical procedure in the United States. Results of analysis of the rate of recurrence of ton-

sillar growth after tonsillectomy indicate that tonsillectomy should command all the respect of a major operation. Reference is made to a report of the Spuths (J. Indiana M. A. 39:401 (Aug.) 1946), who found tonsil stumps in 80 per cent of tonsillectomized patients.

Apparently the writer of this comment is basing much of his information on this report. He states that the usual tonsil stumps are covered by scar tissue, and toxic material is resorbed in its entirety through the lymphatics and veins, and that this may cause fatigue, heart palpitation, vague muscular pain, secondary infections, and allergic complications in remote organs (polyarthritis, nephritis, endocarditis, asthma, chronic phlebitis). If the presence of tonsil stumps cannot be determined by inspection because of scar tissue, their presence can be detected with reliability by eliciting sensitiveness by exerting pressure, either directly on the tonsillar scar tissue or externally from the angle of the lower jaw in the direction of the tonsillar fossa. The writer of the comment then further states that tonsils when functioning normally are protective organs and should not be removed, but that definitely infected tonsils and especially tonsil stumps should be radically removed under general anesthesia and with adequate surgical technique and skill. One wonders in reading this comment if the Spuths were not confusing the lymphoid plaques that often grow, in a small amount, in tonsillar fossae with actual tonsil stumps, inasmuch as they have found such a high incidence (80 per cent) in these 285 tonsillectomized patients who were examined.

BOIES.

LARYNX

Chondroma of the Larynx.

Moore, Paul M.: Cleveland Clinic Quart. 15:4-8 (Jan.) 1948.

In making this report of a chondroma of the larynx in a man 60 years of age, the author directs our attention to the fact that this condition is comparatively rare, only 87 cases, including his own, having been reported to date. He states that in order of frequency it occurs on the endolaryngeal surface of the posterior plate of the cricoid cartilage, the thyroid cartilage, the epiglottis and the arytenoid cartilage. The symptoms of hoarseness, dyspnea and cough are in direct proportion to the location and size of the tumor.

In his case the tumor arose "from the left ala of the thyroid cartilage, presenting a large obstructing mass within the laryngeal

box and a smaller external extension into the neck. The tumor was removed in 1939 by morcellation, using an external approach. A portion of the thyroid cartilage was removed from around the point of attachment. The interior of the larynx was not entered. The patient returned with a recurrence four and one-half years later, at which time the tumor was almost as large as the original one and was again causing signs of laryngeal obstruction. The same method of removal was used, but, in addition, the left ala of the thyroid cartilage was removed. There was a third recurrence three years later, but the mass was quite small and was attached to the skin but not to the larynx. Malignant changes did not develop in this growth. During the seven years that the patient was under observation and treatment he had had normal function of the larynx except for relatively short periods before and after operation. The first recurrence may have been the result of removing too small an amount of thyroid cartilage from around the attachment of the chondroma."

McMAHON.

Surgery of the Larynx and Bilateral Abductor Paralysis.

Kelly, J. D.: *J. A. M. A.* 134:944-946 (July 12) 1947.

This paper consists of a summary and comment on the several modifications of the original King technique, which was introduced for the extralaryngeal approach for the surgical treatment of bilateral abductor paralysis. The paper is concerned mostly with the approach to the exposure of the arytenoid cartilage and whether or not to perform an arytenoidectomy. Kelly's comments are based on a longer experience with this type of surgery than that of any other American laryngologist.

BOIES.

ESOPHAGUS

New Approach to the Treatment of Esophageal Varices.

Som, M. L., and Garlock, J. H.: *J. A. M. A.* 135:628-629 (Nov. 8) 1947.

Two cases are reported in which mediastinotomy was done on patients with severe bleeding from esophageal varices. In the first case, mediastinotomy was necessary after traumatic perforation of the esophagus from endoscopy performed to find the source of esophageal bleeding. The patient recovered and no further bleeding occurred from the esophagus in the six years since he had his mediastinotomy in 1941. It was thought that the trauma from packing stimulated formation of scar tissue and a collateral circulation which cared for

the venous flow and allowed the submucosal veins in the esophagus to atrophy.

Success with the first case encouraged the authors to try a mediastinotomy in another instance of severe bleeding from esophageal varices after injection treatment had failed and the severe bleeding continued. Packing in a superior-posterior mediastinotomy was withdrawn after eleven days and recovery was uneventful. Bleeding occurred five months later but did not persist, and esophagoscopy at that time showed that the upper three-fourths of the esophagus was normal and that there were fibrotic varices around the cardia. The patient had no recurrence of bleeding in the following 14 months, which was as long as he was observed.

BOIES.

Intra-Esophageal Venous Tamponage.

Rowntree, L. G., Zimmerman, E. F., Todd, M. H., and Ajac, J.: J. A. M. A. 135:630 (Nov. 8) 1947.

Confronted with prolonged, uncontrollable, and what was finally believed to be terminal bleeding from the esophagus in a case of cirrhosis of the liver, the authors devised a tampon consisting of a latex bag attached to the distal end of a Miller-Abbott tube, all the perforations of which were occluded except the lower three. The bag was inflated with air, corked, and left in place for several days. The bleeding was controlled and did not recur for two months, at which time the patient was rehospitalized and the tube reinserted. The bleeding was promptly controlled. The tube was removed without incident after 72 hours.

In the second case in which the tube was used bleeding was controlled after the tube had been left in place for 40 hours. The treatment is, of course, probably palliative.

BOIES.

BRONCHI

Emphysema: An Early Roentgen Sign of Bronchogenic Carcinoma.

Rigler, Leo G., and Kelby, Gjert, M.: Radiology 49:578-585 (Nov.) 1947.

The authors describe an early sign obstructive emphysema, in the diagnosis of partial obstruction of a bronchus by a tumor. Five cases are reported in detail and two partially, with 15 roentgenograms to illustrate early and late changes in the lung with bronchogenic carcinoma.

These changes are best demonstrated by roentgenograms or fluoroscopic examinations made during expiration. Transition from emphysema to atelectasis as the tumor grows and completely obstructs the bronchus may be readily determined by repeated roentgen examinations. Patients suspected of bronchogenic carcinoma but without frank roentgen findings should always be examined during expiration as well as inspiration. The finding of minor differences of radiability of the two lungs should lead to further investigation by bronchography, planigraphy, and bronchoscopy.

JORSTAD.

MISCELLANEOUS

Effect of Penicillin on Oral Bacteria.

Long, David A.: Brit. Med. Journal, Nov. 22, 1947.

Many of the organisms which constitute the normal flora of the mouth are penicillin sensitive. Therefore, as Garrod has pointed out, infections derived from or connected with the mouth are in general susceptible to penicillin treatment. McGregor and Long incorporated penicillin in a slowly dissolving pastille for use in the mouth. By renewing these constantly the saliva could be kept bacteriostatic during the day. Changes can be produced in the bacterial flora of the mouth by the administration of penicillin either locally or systemically.

Long treated a total of 500 patients during the years 1944-6 with penicillin administered locally. These were patients with acute gingivostomatitis (Vincent's type), patients with acute and chronic tonsillitis and patients under the care of the maxillofacial and plastic departments.

During the same period he also treated about 30 patients with subacute bacterial endocarditis, severe diphtheria and scarlet fever and actinomycosis with systemic administration of penicillin in doses of 500,000 to 1,000,000 units.

No simple relationship between the penicillin content of the saliva and the penicillin content of the blood seems to exist but Long thinks that fairly adequate amounts of penicillin reach the saliva by excretion during its systemic administration. He thinks further that the threshold for penicillin excretion in the saliva is only obtained by the systemic administration of 500,000 to 1,000,000 units. Furthermore, however large the dose of penicillin administered sys-

temically, penicillin is only demonstrable in the saliva in the initial phases of treatment for as soon as the organisms of the Bacterium group establish themselves penicillinase is produced in sufficient concentration to destroy them.

After the administration of penicillin by either route there was a rapid fall in the number of viable penicillin-sensitive organisms present in the mouth. The changes appeared first in the saliva where the total viable count fell from 60,000,000 organisms per ml. to about 8,000,000 in a few minutes. The changes in the bacterial flora of the gum pockets and the tonsils followed in a few hours and were more striking after systemic administration.

The first phase of rapid reduction of organisms was followed by a second phase of bacterial invasion by organisms of the Bacterium group. Of 312 patients suffering from acute gingivostomatitis of the Vincent's type treated with penicillin pastilles, 71 per cent became infected with gram-negative bacilli between the second and seventh day of treatment and the more conscientious patients became infected with gram-negative bacilli earlier than did the careless ones. All patients treated systemically with a million units per day for more than three days developed a secondary bacillary infection on the third or fourth day of treatment. Once present these invading organisms multiplied rapidly in the saliva until the count of viable organisms was about 60,000,000 per ml. On stopping the treatment they rapidly decreased in numbers as the normal flora reestablished itself.

GROVE.

Penicillin Inhalation Therapy.

Segal, M. S., Levinson, L., and Miller, D.: J. A. M. A. 134:763-770 (June 28) 1947.

The authors review an experience with penicillin inhalation therapy in the treatment of pneumonia, infective bronchial asthma, bronchiectasis, sinusitis, pulmonary abscess, acute laryngotracheobronchial edema, and chronic pulmonary emphysema.

Studies were made to determine whether penicillin actually found its way into the sinuses by way of the ostia or whether it was brought to the sinuses through other channels. Radiopaque aqueous substances such as "skiodan" and "diodrast" ranging in concentration from 10 per cent to 60 per cent were aerosolized in the nasal passages. Immediate follow-up roentgenograms showed that the ra-

diopaque material had not found its way into the sinuses. This seemed to imply that penicillin gets into the sinuses by way of the veins and lymphatics in the mucosal lining of the nose rather than by way of the ostia.

In some of the studies, 16,000 units of penicillin and 2.0 cc. of isotonic solution of sodium chloride were given every three hours. In others, the amount of penicillin was increased to 25,000 units. One-fourth per cent neo-synephrine hydrochloride was used as a shrinking agent before treatment in most cases.

Of the 18 cases which were studied clinically and by roentgen rays, there were nine cases of chronic sinusitis complicated by bronchiectasis, five cases of chronic sinusitis uncomplicated by pulmonary disease, and four cases of acute sinusitis. Only one case history is included. This indicates a rather remarkable result in a 19-year-old patient with bilateral chronic pansinusitis with subacute exacerbation. Penicillin aerosol therapy was given for four days. The details regarding the other cases are lacking; this obviously detracts from the value of this report.

Six patients with acute laryngotracheobronchial edema were treated with penicillin aerosol. (These do not seem to be cases of laryngotracheobronchitis, which most laryngologists regard as a clinical entity.) The only case report is that of a 46-year-old man who failed to respond to parenteral use of penicillin, oxygen, etc., but on whom the use of penicillin aerosol with a tracheotomy tube and a flow of 75 per cent helium and 25 per cent oxygen apparently produced dramatic results and prompt recovery. The ages of the other patients are not mentioned.

BOIES.

Aerosol Penicillin.

Prigal, S. J., McGavack, T. H., Speer, F. D., and Harris, R.: J. A. M. A. 134:931-938 (July 12) 1947.

This paper is concerned with the blood levels of penicillin obtained by inhalation of aerosols produced by a combined steam generator and aerosolizer with the use of propylene glycol in tents and through a breathing box. Otolaryngologists will be particularly interested in their description of a portable translucent plastic tent for the administration of aerosol penicillin in propylene glycol; this medication should be particularly useful in the treatment of acute obstructive laryngitis in children. The humidity and the temperature

can easily be controlled and unusually high and prolonged levels of penicillin can be obtained in the blood when a penicillin-propylene glycol aerosol is inhaled within the tent. Thus repeated injections can be avoided. This paper should be read in its entirety by anyone concerned with the treatment of acute obstructive laryngitis.

BOIES.

"Liquid" Versus "Solid" Penicillin in Oil and Wax.

Dowling, H. M., Romansky, M. J., et al.: *J. A. M. A.* 135:567-569 (Nov. 1) 1947.

This is a study of the effect of particle size and type of penicillin on the concentration of the drug in the blood. Preparations of crystalline penicillin in oil and wax were found to be superior to preparations which were comparable in other respects but which contained amorphous penicillin. Solid preparations gave optimal results if crystalline penicillin was used, whether the particles were of large or small size, or if amorphous penicillin was used when the particles were of large size. Liquid preparations gave optimal results if crystalline penicillin of large particle size was used and almost as good results with amorphous penicillin of large particle size. Liquid preparations containing penicillin of small particle size, whether amorphous or crystalline, gave definitely inferior results. On the basis of ease of administration as well as on the concentration of penicillin achieved in the blood, the liquid preparations of crystalline penicillin of large particle size are recommended as the most satisfactory form of penicillin in oil and wax.

BOIES.

Tissue Changes in Allergy.

Kline, B. S.: *J. Allergy* 19:19-33 (Jan.) 1948.

The observations from a study of routine microscopic examination of surgical and autopsy specimens over a 12-year period by the authors and of similar studies recorded in the literature are clearly summarized:

"1. As suggested by Doerr, the term 'allergy' is used to denote all disorders due to antigen-antibody reaction including those of atopy, idiosyncrasy and anaphylaxis.

"2. Characteristic of allergic reactions are rapid onset, violent course and slow regression.

"3. The tissue changes in allergy vary from no detectable morphologic alteration of cells to inflammatory, proliferative, severe degenerative, and necrotic lesions.

"4. In general, the tissue changes in allergy reflect the severity of the signs and symptoms of the reactions. This is not invariably the case, however, as is evidenced by the finding of no appreciable morphologic change of sensitized smooth muscle in animals dead from anaphylactic shock.

"5. It is probable that antigens possess a variable capacity to sensitize and shock different tissues of the body.

"6. Since nonsensitized and sensitized tissues may respond to different foreign substances in the same manner and, on the other hand, react in a different manner to the same antigen or happen on second or subsequent exposure, a complete understanding of the tissue changes, therefore, requires a knowledge of the state of the tissues when injured no less than a knowledge of the inherent toxicity of the foreign excitant."

In the light of current practices the following statement is particularly significant. "The most dangerous of all types of hypersensitivity apparently is that of idiosyncrasy in which, for instance, sulfonamide or other drug conjugates with the body's own plasma protein and sensitizes most of the body's tissues including the parenchymatous organs."

It is this principle which definitely contraindicates the casual and repeated administration of the sulfonamides and antibiotics, in prophylaxis as well as in therapeusis.

McMAHON.

Diphenhydramine Hydrochloride ("Benadryl Hydrochloride").

Blumenthal, L. S., and Rosenberg, M. H.: J. A. M. A. 135:20-24 (Sept. 6) 1947.

A report is made on the use of this drug in 137 patients. A large number of different conditions were treated. No attempt is made at evaluation in the smaller groups of patients, such as in the three cases of vasomotor rhinitis in which one received pronounced relief and the others only moderate relief. The best results were obtained in the treatment of urticaria and hay fever. Its use in bronchial asthma was not encouraging. The most important part of this report deals with the intravenous use of the drug in 16 cases. Twenty to 50 mg. in 75 to 100 cc. of isotonic sodium chloride solu-

tion was administered by intravenous drip in approximately ten minutes. Eleven patients with urticaria were treated by this method and ten had complete cessation of itching at the completion of the infusion. In the two cases of bronchial asthma there was no improvement. The administration of the drug intravenously eliminates other factors in deciding whether the drug is effective in a specific condition. It is considered that this method of administration is an aid to the establishment of indications for use of the drug in those conditions in which its efficacy is equivocal.

BOIES.

A Clinical Test For Migraine.

Schnitker, M. T., and Schnitker, M. A.: J. A. M. A. 135:89 (Sept. 13) 1947.

The authors have found that the sublingual administration of nitroglycerin (1/50 grain or 1.3 milligrams) in the form of soluble tablets is a highly satisfactory method for the experimental production of migraine headache in persons who are susceptible. They have used it in over 250 cases. In those cases in which the nitroglycerin test has reproduced the headache, good results have been obtained by treating the patient for migraine. This treatment consists chiefly in the administration of 10 grains (0.65 grams) of calcium lactate twice a day, the use of a sedative such as ½ grain (0.03 grams) of phenobarbital twice a day, the elimination of allergenic substances when they can be discovered (particularly coffee, chocolate, beans, peanuts, and coca cola) and the use of ergotamine tartrate, 2 to 4 milligrams under the tongue at the onset of an acute attack.

The authors refer to the work of Wolff in determining that the basic mechanism seen in the migraine syndrome is due to stretching of the cranial arteries, chiefly the extracranial structures (external carotid artery and branches). They state that this same mechanism is seen in the headache associated with arterial hypertension; conversely, the headache of histaminic cephalgia and that associated with fever are due chiefly to dilatation of the cerebral branches of the internal carotid, the vertebral or the basilar arteries. One of the authors, in 1938 while working with Northfield, demonstrated that the principal action of histamine in producing headache is on the internal carotid artery and its branches.

Horton's test is referred to, in which a diagnosis of histaminic cephalgia is made when histamine is given subcutaneously and the patient's headache is reproduced.

BOIES.

Cavernous Sinus Thrombosis.

Fox, F. L., and West, G. B., Jr.: J. A. M. A. 134:1452-1456 (Aug. 23) 1947.

Newer concepts in the management of this grave disease are reviewed and three cases are reported; two of the patients recovered (one with blindness). The authors thoroughly review the etiology and the diagnosis. They strongly advise the combined use of sulfonamide compounds, antibiotics, and the anticoagulants in large doses. They believe that if the case is unresponsive to one of the sulfonamide drugs, another should be substituted. They suggest that the same is probably true of antibiotic agents. They also believe that the early employment of heparin and "dicoumarol" administered simultaneously is desirable.

BOIES.

Books Received

Hearing and Deafness: A Guide for Laymen.

Edited by Hallowell Davis, M.D., Research Professor of Otolaryngology and Associate Professor of Physiology, Washington University Medical School; Director of Research, Central Institute for the Deaf. Pp. xv+496, illustrated. New York, Murray Hill Books, 1947. (Price $5.00)

This is one of the most concise and at the same time comprehensive works on the subject. It will no doubt prove to be even more useful to otolaryngologists and to teachers than to laymen. Owing to the nature of the subject it is difficult to avoid technicalities which even in their simplest form will be difficult for the untrained layman. Sections of the book are devoted to physics and psychology of hearing, the medical and surgical aspects of treatment, and the testing of hearing.

Later chapters, of more special concern to the layman, deal with hearing aids, their choice and management, and with the adaptation of the patient to his condition and his environment.

In our opinion, one of the important books of the year.

Synopsis of Allergy.

By Harry L. Alexander, A.B., M.D., Professor of Clinical Medicine, Washington University School of Medicine, St. Louis; Editor of The Journal of Allergy. 2nd ed., pp. 255, with 22 illustrations. St. Louis, The C. V. Mosby Company, 1947.

Alexander's book is a small volume, truly a synopsis, complete and concise in its content and well illustrated.

It should prove a useful reference book for the otolaryngologist intent on keeping abreast of this rapidly changing subject and confused by the voluminous literature.

Headache.

By Louis G. Moench, M.D., Assistant Clinical Professor of Medicine, University of Utah School of Medicine; Internist, Salt Lake Clinic, Salt Lake City. Pp. 207 with 59 illustrations. Chicago, The Year Book Publishers, 1947. (Price $3.50)

Within the small compass of 200 pages the author has managed a complete and very informative outline of the literature of headache. He approaches the subject from the standpoint of origins,

devoting a chapter to each type: headaches from intracranial disturbances, neuralgias, ocular and nasal headaches, those resulting from pathology in the neck, from systemic and emotional aberrations, from histamine, and finally migraine. The descriptions are clear and the illustrations excellent.

Ear, Nose and Throat. Symptoms, Diagnosis, Treatment.

By George D. Wolf, M.D., Assistant Clinical Professor of Otolaryngology, New York Medical College. Pp. xx+523, with 149 illustrations, including 25 in color. Philadelphia, J. B. Lippincott Co., 1947. (Price $10.00)

"Designed for use in general office practice," states the blurb on the jacket, "and for all who desire a wider comprehension of the subject, yet do not wish to become enmeshed in a welter of details."

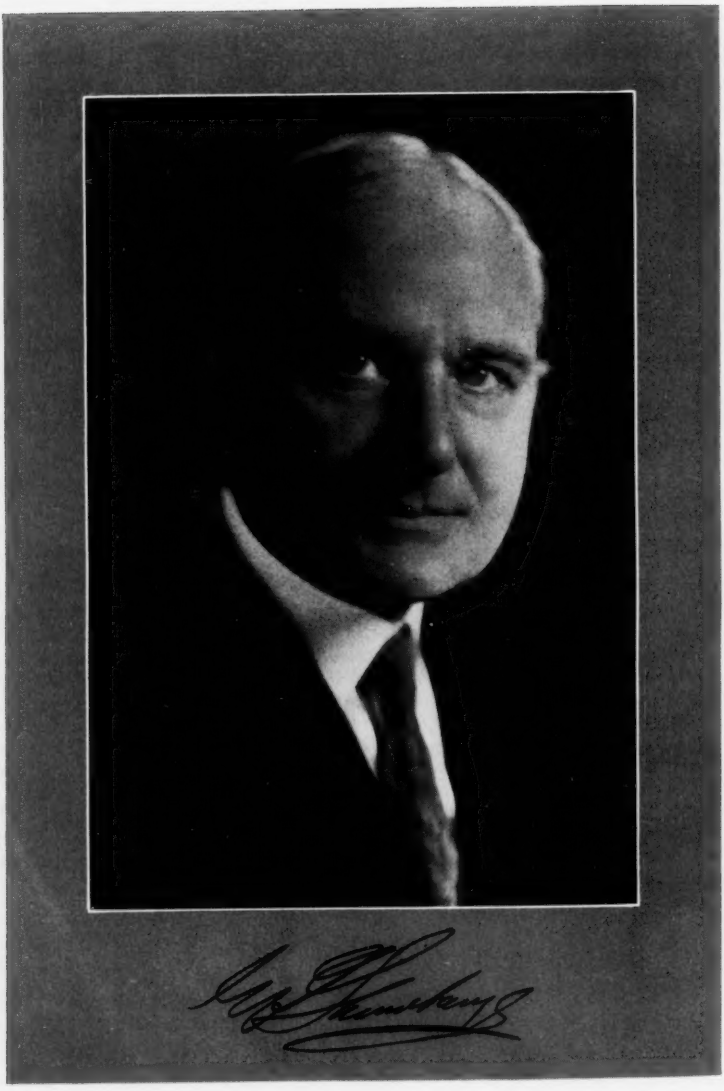
It is our impression that the author would have done well to enmesh himself somewhat deeper in details before essaying a teaching volume.

Superficial and none too accurate.

Endotracheal Anesthesia.

By Noel A. Gillespie, D.M., B.Ch., M.A. (Oxon.); M.D. (Wis.); D.A. (R.C.S. Eng.); Associate Professor of Anaesthesia in the University of Wisconsin; Sometime: Senior Resident Anaesthetist, The London Hospital; Hon. Assistant Anaesthetist, The London Hospital; Hon. Assistant Instructor in Anaesthetics, London Hospital Medical College and Dental School; Hon. Anaesthetist, The Connaught Hospital, Walthamstow, Anaesthetist to the Plastic Department, Princess Elizabeth of York Hospital for Children, Shadwell; Research Associate and Resident in Anaesthesia, University of Wisconsin, State of Wisconsin General Hospital. Second edition. Pp. xvii+237, illustrated. Madison, The University of Wisconsin Press, 1948. (Price $4.00).

This is the second edition of a very useful manual on anesthesia. It is well adapted as a reference work not only for anesthetists but for surgeons and the surgical specialists.



GEORGE E. SHAMBAUGH, M.D.

1869-1947

With the death of Dr. George E. Shambaugh on November 30, 1947, otolaryngology lost one of its most honored members. Dr. Shambaugh was born in Clinton County, Iowa, November 15, 1869. He received the degree of Bachelor of Philosophy at the University of Iowa in 1892, and the degree of Doctor of Medicine at the University of Pennsylvania in 1895. Following this, Dr. Shambaugh spent one year at the University of Berlin and a second at the University of Vienna. On May 2, 1901, he married Edith Capps of Jacksonville, Illinois.

Dr. Shambaugh became Professor of Otolaryngology and was Chairman of the Department at Rush Medical College from 1916 until his retirement in 1935. He was Otologist and Laryngologist at the Presbyterian Hospital from 1902 until 1935. In 1918, Dr. Shambaugh organized a one year basic post-graduate course in otolaryngology at Rush Medical College. From eight to twelve men were trained each year during the period from 1918 to the start of World War II.

In 1912, the Baron de Lenval prize for research on the internal ear was awarded to Dr. Shambaugh. He was a contributor to the Annals of Otology, Rhinology, and Laryngology, Journal of the American Medical Association, several foreign journals, and was Editor-in-Chief of the Archives of Otolaryngology from its inception until 1937. Dr. Shambaugh wrote chapters on the ear and nasal sinuses for several textbooks. He was for many years editor of the Year Book Company, and from 1931 to 1937 was sole editor of the Ear, Nose and Throat Section.

Dr. Shambaugh was a Fellow of the American Otological Society and past president, Fellow of the American Laryngological Association and past president, a member of the Chicago Laryngological and Otological Society, being president in 1910, a member of the American Academy of Ophthalmology and Otolaryngology, and of the American Laryngological, Rhinological, and Otological Society, The American Medical Association, The Chicago Medical Society, and the Institute of Medicine of which he was also past president. He was Chairman of the Section of Otolaryngology of the American Medical Association 1911-1912, and a member of the

House of Delegates in 1916. He was the organizer and founder of the American Board of Otolaryngology.

Dr. Shambaugh was internationally known for his research on the ear, especially that of the blood supply and histological details of the inner ear. His ability to distinguish the important from the unimportant, and to speak and write clearly and to the point, will long be remembered by those who were fortunate enough to have known him.

E. W. HAGENS.

Notices

UNIVERSITY OF MINNESOTA

The University of Minnesota announces the 6th biennial Continuation Course in Otolaryngology to be held June 28, 29, 30, July 1, and 2. This Course is designed to bring to the practicing otolaryngologist the newer concepts and developments in the specialty. The Course will be under the direction of Dr. Lawrence R. Boies and associates of the University Medical School. Dr. Harold I. Lillie, Dr. Gordon B. New, and others of the Graduate School Faculty will participate in the instruction. Guest lecturers will include Dr. Kenneth Day, Pittsburgh, Dr. Gordon Hoople, Syracuse; Dr. C. Stewart Nash, Rochester, N. Y.; and Dr. Francis LeJeune, New Orleans.

The fee for this Course is $35.00. The enrollment is limited. Application should be made at an early date to the Director, Center for Continuation Study, University of Minnesota, Minneapolis 14, Minnesota.

OFFICERS

OF THE

NATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: Dr. Carl H. McCaskey, 608 Guaranty Bldg., Indianapolis 4, Ind.
President-Elect: Dr. Conrad Berens, 35 East 70th St., New York, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 10-15, 1948.

American Broncho-Esophagological Association

President: Dr. Millard F. Arbuckle, 539 N. Grand Ave., St. Louis 3, Mo.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11, Ill.
Meeting: Chalfonte-Haddon Hall, Atlantic City, N. J., April 7-8, 1948.

American Laryngological Association

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis 8, Mo.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: The Homestead, Virginia Hot Springs, April 14-15, 1948.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lyman G. Richards, 1101 Beacon St., Brookline, Mass.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Meeting: Chalfonte Haddon Hall, Atlantic City, N. J., April 7, 8, 9, 1948.

Sections:

Eastern—Chairman: Dr. J. Winston Fowlkes, 12 East 63rd St., New York 21, N. Y.

Southern—Chairman: Dr. William A. Wagner, American Bank Bldg., New Orleans 12, La.

Middle—Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.

Western—Chairman: Dr. Meade Mohun, 23 Second Ave., San Mateo, Calif.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Secretary: Dr. James M. Robb, 641 David Whitney Bldg., Detroit, Mich.
Meeting: Chicago, Ill., June 21-25, 1948.

American Otological Society

President: Dr. Bernard J. McMahon, 634 N. Grand Ave., St. Louis 3, Mo.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.
Meeting: The Homestead, Virginia Hot Springs, April 12-13, 1948.